Journal

OF THE

AMERICAN VETERINARY MEDICAL ASSOCIATION

Integrated Livestock Production

A REPORT—what integration can—and cannot—do. Page 95

Rational Disease Control Programs

EPIZOOTIOLOGY—not a problem for the research man, alone, but also for the alert veterinarian. Page 97

Routine Pregnancy Examination of Cattle

A SERVICE increasingly in demand by owners of beef cattle. Page 130

Ovine Virus Abortion

A STUDY of the causes of virus abortion in sheep in Montana. Page 133

Vol. 136 February 1, 1960 No. 3

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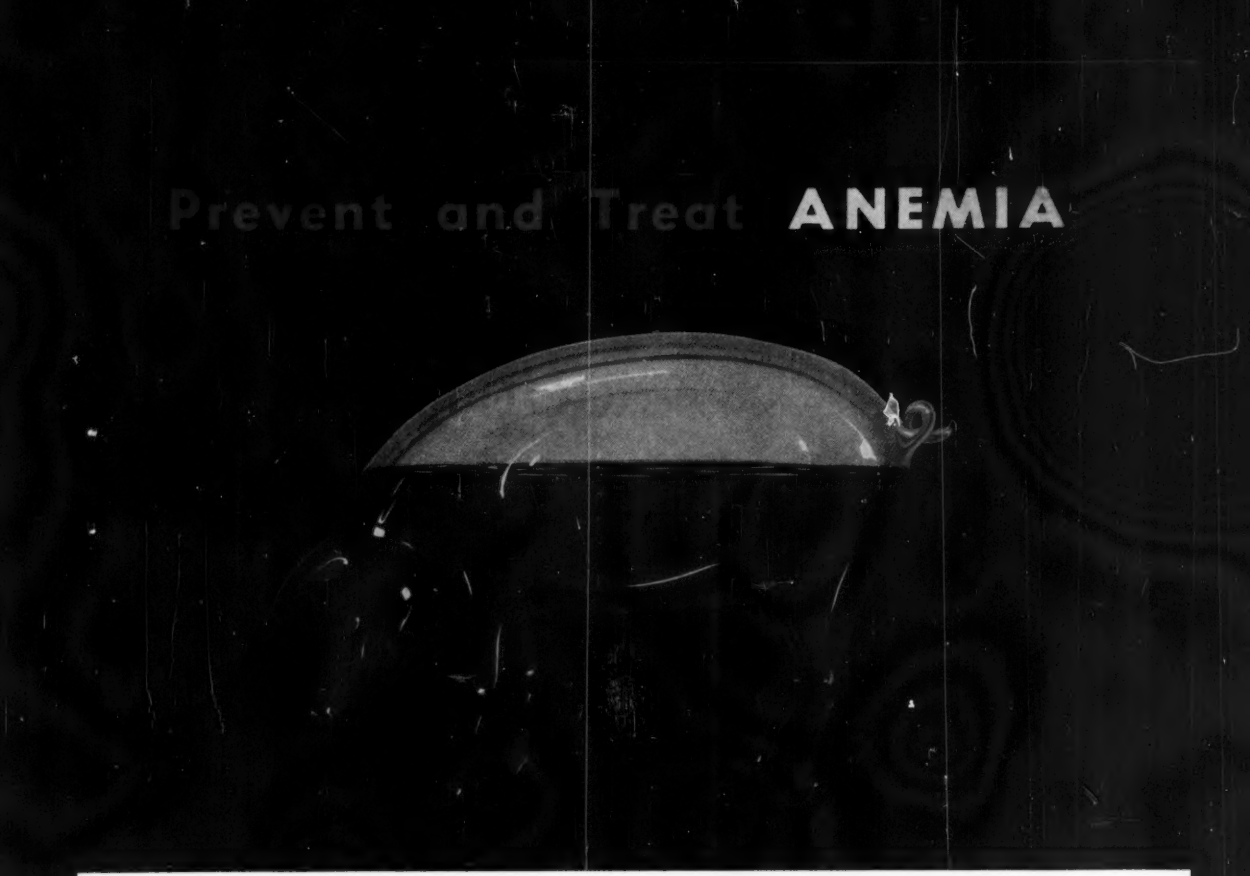

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Journal

OF THE
AMERICAN VETERINARY
MEDICAL ASSOCIATION

Vol. 136 No. 3 Feb. 1, 1960

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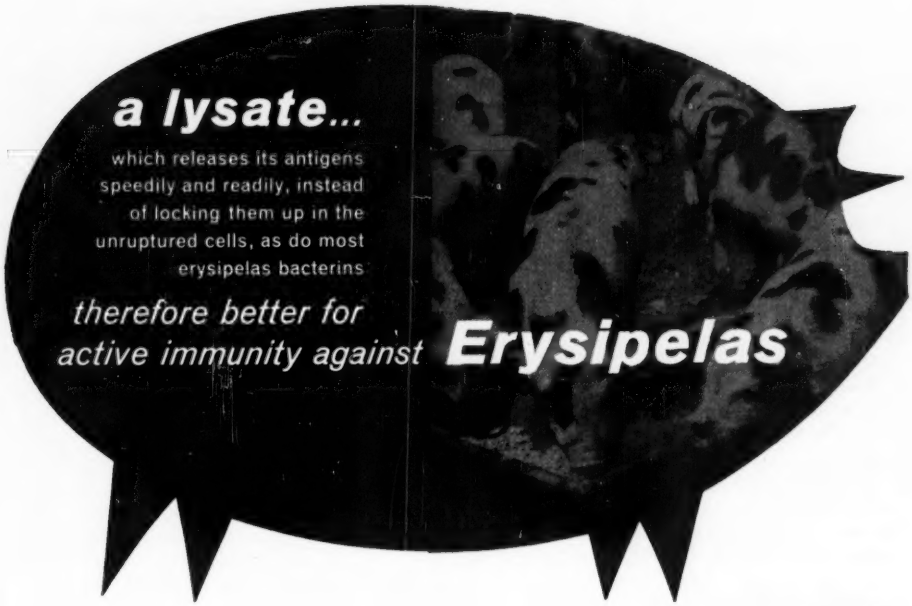
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Correspondence

December 15, 1959

Dear Sir:

It is a pity to see an elegant paper like Jensen's "Hypopituitarism Associated with Cystic Rathke's Cleft in a Dog" (JOURNAL, Dec. 1, 1959: 572) published with four references to man and laboratory animals but none to dogs. Jensen states: "A review of the veterinary literature indicates that hypopituitarism in the dog as a result of cystic formation of Rathke's cleft is relatively rare." This statement is conservative enough to be innocuous, but leaves the reader wondering, how rare?

One can hardly write on this subject—especially if one is going to cite what goes on in man—without referring to Oboussier's classic studies on the dog. In an exhaustive treatise (Die Rachendachhypophyse des Hundes, *Zeitschr. f. wiss. Zool.*, 156, 1944: 365-438) she described the pharyngeal hypophysis of the dog and the anomalies that occur in the hypophyseal region amongst various breeds of dogs. Cysts lined with ciliated epithelium are mentioned by Kingsbury and Roemer, whose paper (The Development of the Hypophysis of the Dog, *Am. J. Anat.*, 66, 1940: 449-481) should not be overlooked by anyone interested in the pituitary of this species. An author reporting a single case of a disease is, of course, under no obligation to cite any literature at all; but if he does, it should at least be pertinent to the species with which he is dealing.

At least one reference to a clinical case of hypopituitarism in a dog would have been desirable. A similar case, also in a German Shepherd Dog of about the same age (11 mo.), was reported by Moch and Haase (Hypofunktion der Adenohyohphyse eines Hundes, *Tierarztl. Umschau*, 1953: 242). A cyst which had replaced most of the hypophysis was also present in their dog.

Sincerely yours,

s/LEON Z. SAUNDERS, D.V.M.

Philadelphia, Pa.

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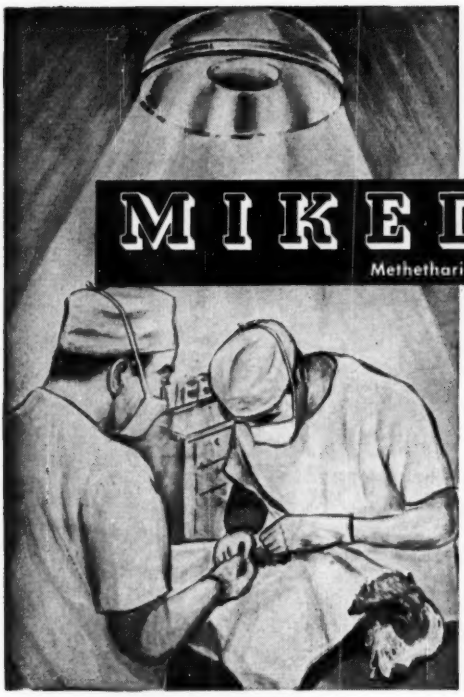

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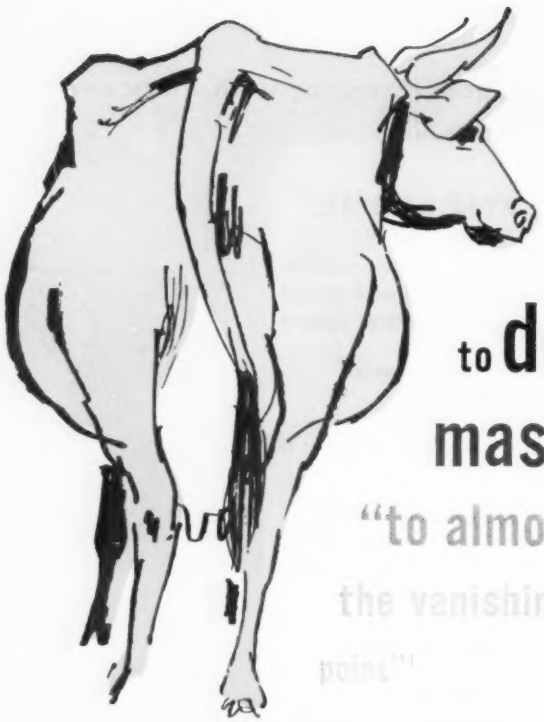

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1. Kakavas, J. C., et al.: J. Am. Vet. M. Ass. 119:203 (Sept.) 1951.

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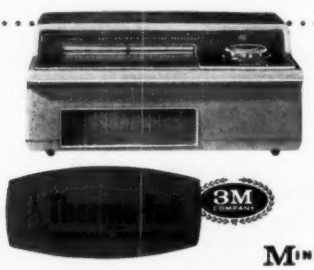

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FROM THE AVMA WASHINGTON OFFICE

LEGISLATIVE ITEMS

Changes in FDA Laws Introduced

According to John L. Harvey, deputy commissioner of the Food and Drug Administration, there are five major types of legislation that would make substantive changes in laws administered by the F.D.A. This legislation pertains to (1) barbiturates and amphetamines, (2) color additives in or on foods, drugs, or cosmetics, (3) pre-testing of cosmetics (would apply to same general control now applied to new drugs, food additives, and pesticide chemicals), (4) pre-clearance of new medicinal devices, would require therapeutic devices tested for safety and results of tests accepted by F.D.A. before devices could be marketed commercially (would apply same control to new devices that the present law applies to new drugs), and (5) labeling warnings on hazardous substances for household use and, where necessary, adequate directions for firstaid treatment. The Department of Health, Education, and Welfare has drafted a bill pertaining to therapeutic devices, and bills in each of the other areas have been introduced in the 86th Congress.

MISCELLANEOUS

Research Grant Awarded to Univ. of Penn.

Dr. I. S. Ravdin, vice-president for Medical Affairs, University of Pennsylvania, announced that National Heart Institute, U.S.P.H.S., awarded a $1,100,000 grant to the Univ. of Penn. school of veterinary medicine for ten years of research in comparative cardiovascular diseases of man and animal. Dr. David K. Detweiler, associate professor of veterinary pharmacology and chief of the laboratory of physiology and pharmacology, school of veterinary medicine, will serve as director and principal investigator of the research effort. He will establish the Univ. of Penn. Comparative Cardiovascular Studies Unit (CCSU), which will serve as the research center for the project. The unit will also carry out an international training program for research in comparative cardiology of the World Health Organization.

W.H.O. May Implement Medical Exchange

W. H. O. executive board, which meets in Geneva this month, is expected to help implement a new medical exchange agreement between the Soviet Union and the United States. The two-year bilateral pact contemplates co-sponsorship of scientific investigations, interchange of study teams, mutual support of medical literature and films, and exchange of medical or public health exhibits.

A.E.C. Awards 37 Equipment Grants

The Atomic Energy Commission has awarded grants for bio-nuclear training to 36 medical schools, colleges, and universities. Grants are for acquisition of equipment needed in radiation biology studies in medical, para medical and biological disciplines. These grants amounted to $434,601 and bring the total sum awarded under this special program to $2,627,406 since it was introduced in October, 1957.

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No, this won't happen overnight. It's a slow, eating-away process. That is why it's such an important issue—and why we call it to your attention. We think your interest in this matter is the same as ours.

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1. Harris, J. R. and Clarkson, T. B.: Prevention of Relapses in Milk Fever, Vet. Med. 12:696 (Dec., 1955).

2. Baker, W. L., Jr.: Prevention of Relapses in Milk Fever, N. Am. Vet., 37:203 (Mar., 1956).

3. Graham, K.D.: Milk Fever Relapses, Vet. News (Canada) 20: (Nov., 1958).

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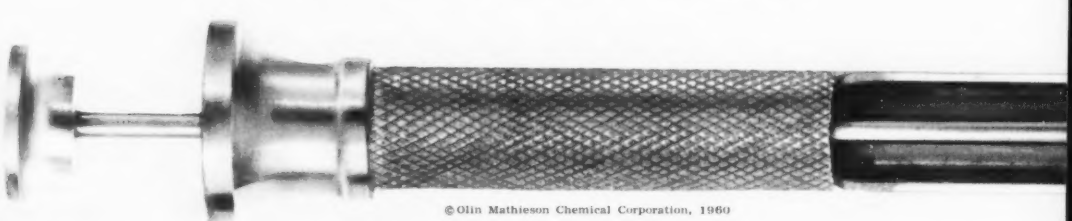

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SYNOVEX-S contains 200 mg. of progesterone and 20 mg. of estradiol benzoate. Contents of entire 1-dose cartridge (8 tiny pellets) are implanted simultaneously. Recommended for steers weighing from 400 to 1000 lbs. One implant lasts entire finishing period.

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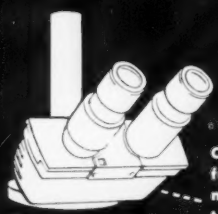

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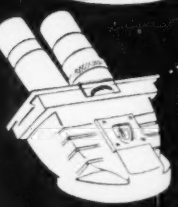

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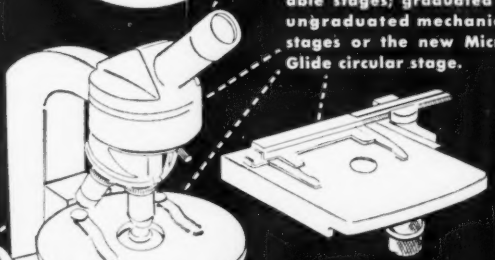

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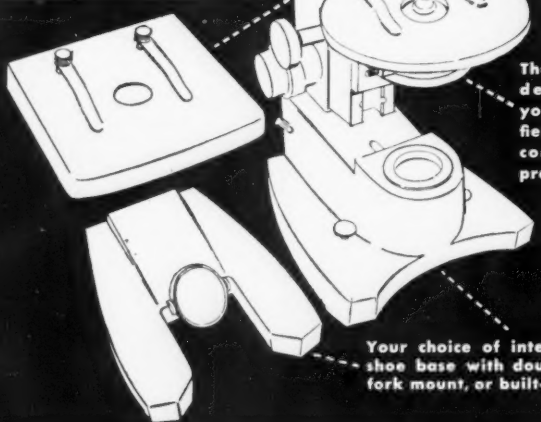

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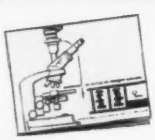

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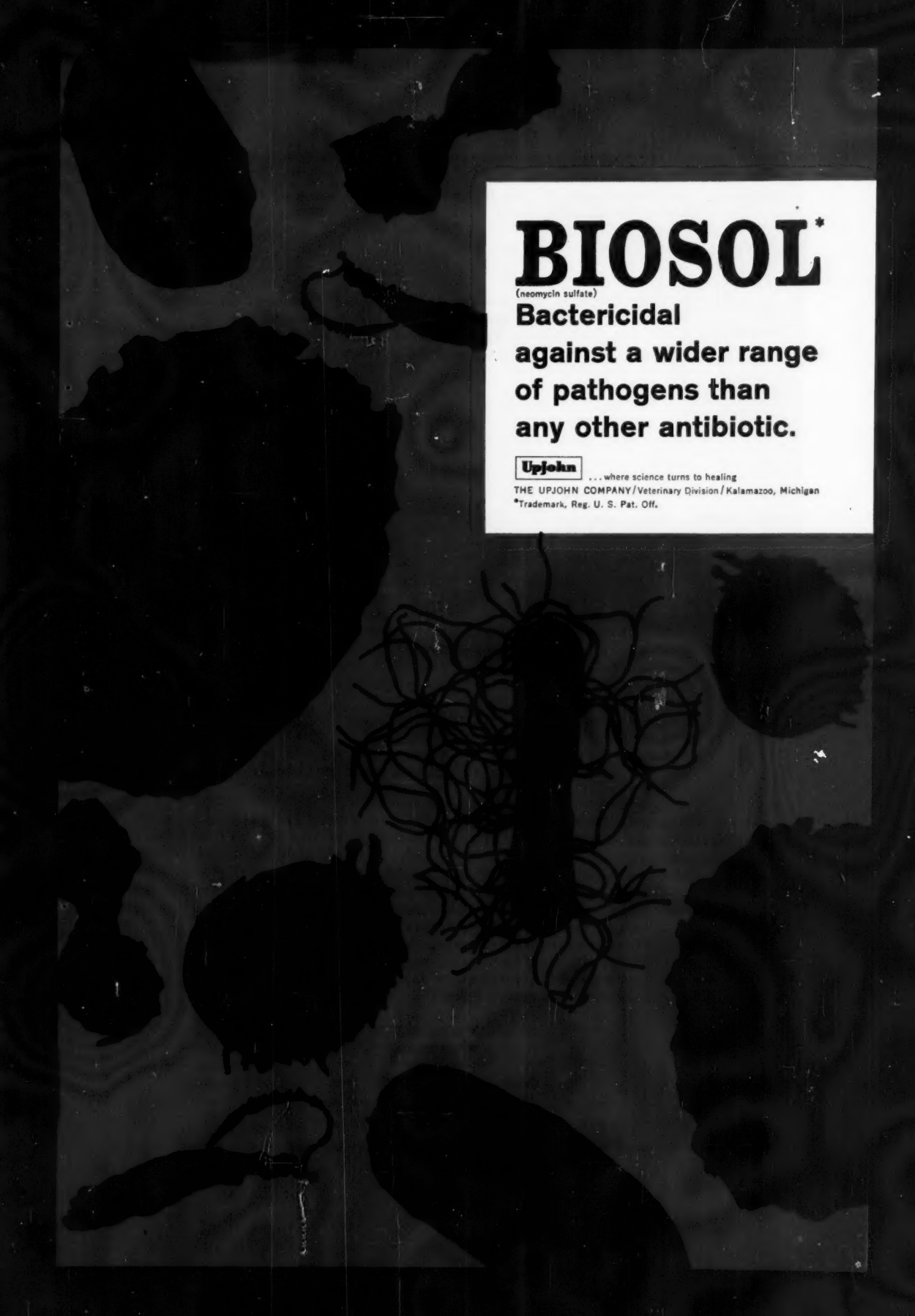
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To: Doctors of Veterinary Medicine

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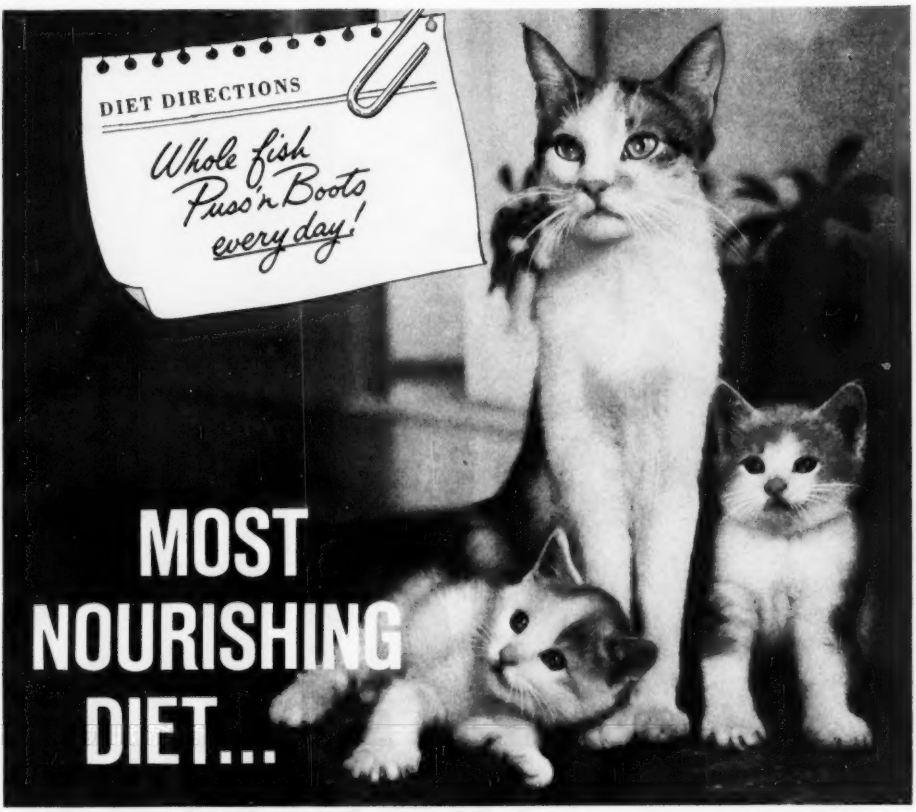

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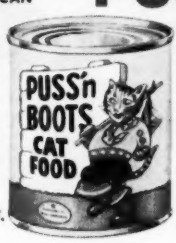

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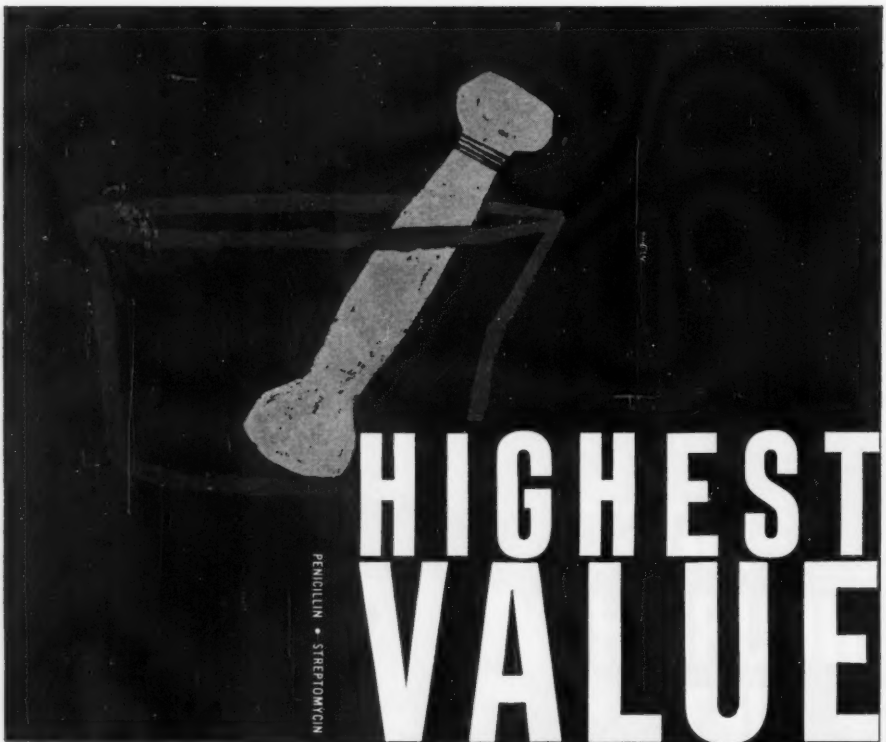

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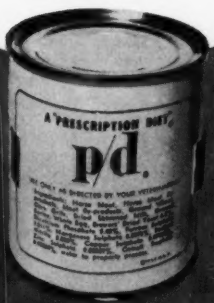

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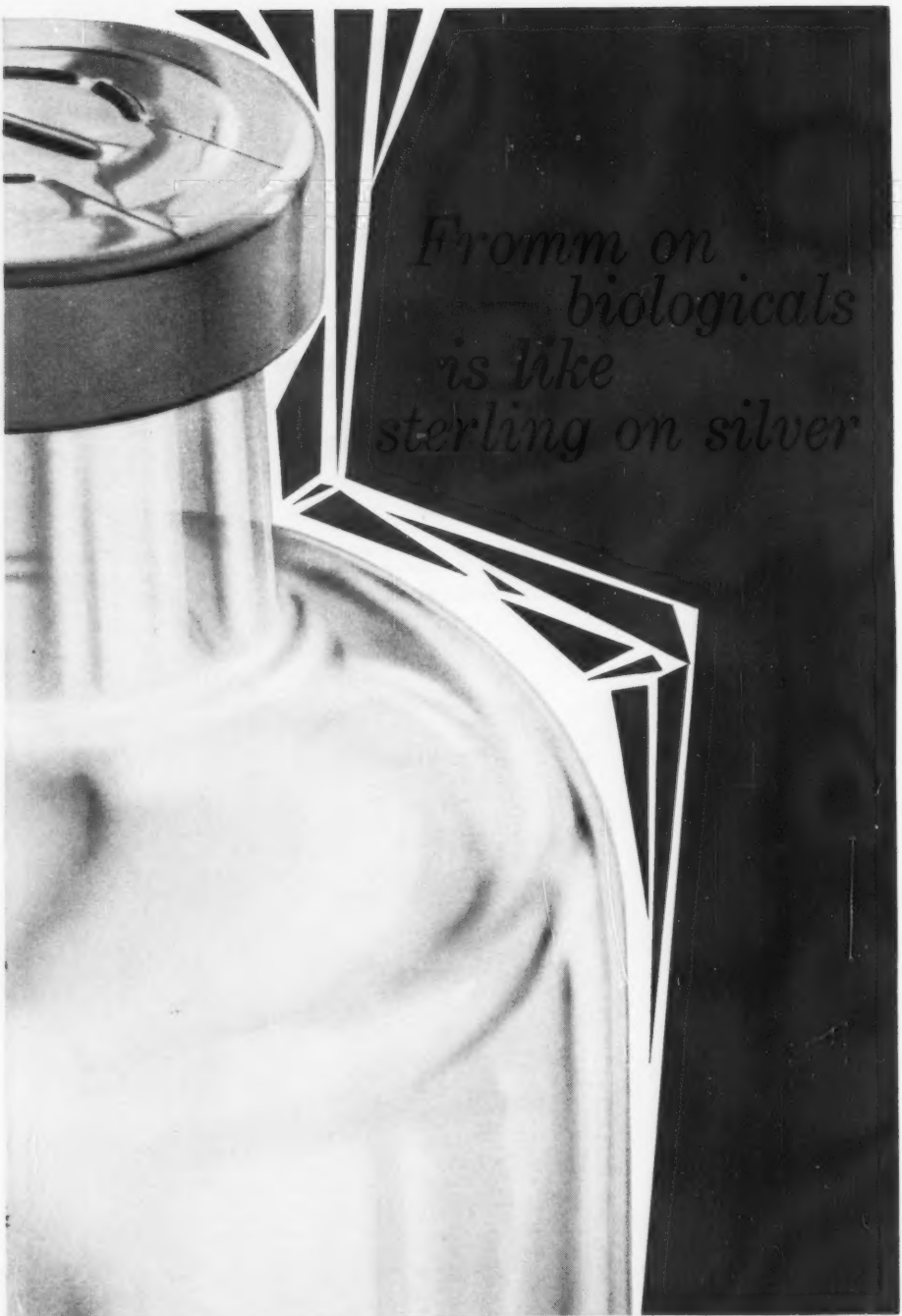

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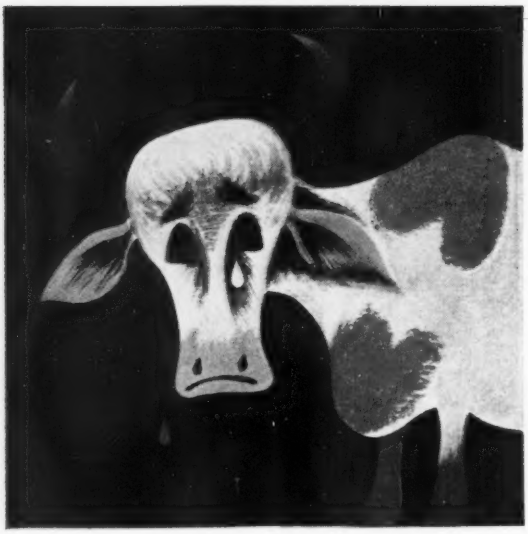

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- **reduces services per conception**
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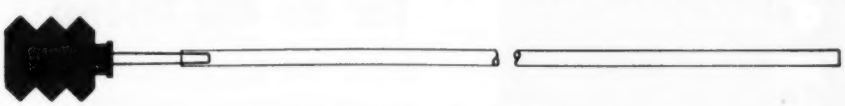
Impaired fertility in cows, commonly accepted to result from non-specific genital infection, responds dramatically to the 2-step treatment method with FURACIN. In one 3-year study involving treatment with FURACIN Suppositories Veterinary, of approximately one-half of 530 "problem breeders": "The average number of services per conception was 1.88 in the treated and 2.85 in the untreated cows. The average number of days between calvings was 385 in the treated and 447 in the untreated animals."[1]

In a study with 85 subfertile dairy cows, 61.7% of 34 treated with an intra-uterine injection of FURACIN Solution Veterinary conceived at first service, while only 19.3% of 31 treated in an identical manner with 10% saline solution conceived at first service; 40% of 20 non-treated controls conceived at first service.[2]

FURACIN 2-Step Method for Impaired Fertility

STEP 1. During estrus, instill, aseptically, the contents of 1 to 3 SQUEEJETS (30 to 90 cc.) of FURACIN Solution Veterinary into the uterus by means of a uterine pipette.



STEP 2. During the following 3 weeks, insert 1 FURACIN Suppository Veterinary into the anterior portion of the vagina 3 times each week on alternate days.

The cow may then be bred during the next estrus.

Supply: FURACIN Solution Veterinary SQUEEJET (30 cc. each), boxes of 12; FURACIN Suppositories Veterinary, boxes of 12.

1. Vigue, R. F., et al.: J. Am. Vet. M. Ass. 134:308 (April 1) 1959.
2. Vigue, R. F.: Personal communication.

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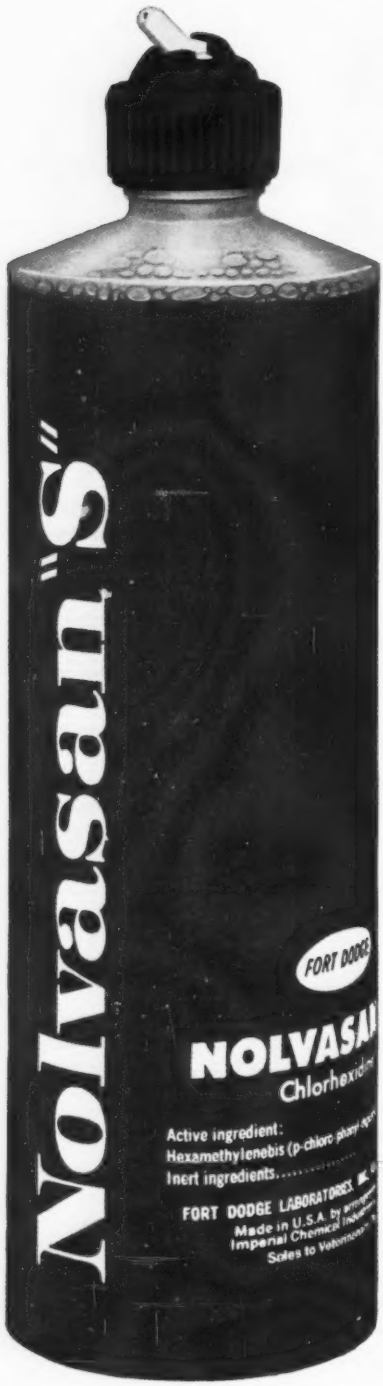

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Vol. 136

No. 3

February 1, 1960

Journal

OF THE AMERICAN VETERINARY MEDICAL ASSOCIATION

Integrated Livestock Production

R. L. COPPERSMITH, PH.D.

IN THE PAST few years a popular topic in the livestock business has been "integration." It is a much maligned term used to describe about everything describable and a few things that are not. Integration may be complete or partial—it may involve companies, corporations, or one-man firms. It may involve contracts between various segments of an organization; it may involve total ownership of all the segments necessary to create a product and move it through the channels of trade to the consumer, the whole process being supervised and managed by one controlling group. It may be voluntary or compulsory. It can lead to more efficiency or it can lead to less efficiency. It may lead to a loss of freedom or it may lead to an increase in the amount of freedom individuals in a given situation may have. It is all based on the great battle to influence the minds of men; to control land, labor, capital, and management; to produce more of what people want, when they want it, in the place they want it, in the form they want it, and at a price they can afford to pay.

What Integration Can Do

Integration may, and usually does, lead to an increase in the size of the economic unit in the livestock industry. Up to a certain point, this will, in general, lead to more efficiency, *i.e.*, less overhead per pig or calf, or less fixed expense per unit of production. If the efficiency is carried all the way through to the final consumer, it may lead to a lower price per unit bought and more satisfaction for the consumer per dollar. If so, it affects the well-being of 175,000,000 people. If we get more value per consumer dollar, then who can say that integration is bad, for anything that increases the well-being of 175,000,000 people cannot be argued against for very long.

In increasing the size of business, integration may lead to the use, or concentrate the use, of new technologic methods, since the "5 sows and 2 cows man" is not likely

---

Based on a presentation given at the Veterinary Short Course, sponsored by the College of Veterinary Medicine and Illinois Veterinary Association, Oct. 15-16, 1959, Urbana, Ill.

Dr. Coppersmith, Decatur, Ill., head of the feeder pig department of the Illinois Livestock Marketing Association, is now extension economist at Kansas State University Manhattan

to be in a position to know about new techniques or to be financially able to use them even if he knew about them. In using new technologic methods, we may get a better quality product to satisfy the American consumer.

Integrated livestock production may lead to more price stability through more stable supplies of livestock. For years, the American meat industry has experienced difficulties due to instability in livestock numbers. There are fluctuations in the quality and quantity of the products coming on the animal market at different times. This has made it difficult for American food merchandisers to lay out their products when and where the customer wants them and in the amount and quality desired. If integration leads to more stability in amounts and qualities of products, we may see less erratic price variation.

An integrated system may enable us to concentrate research, education, and promotion efforts to benefit the livestock industry. Consequently, we may come up with new products, better technologic methods, and better ways to motivate human minds to get them to buy our products.

What Integration Cannot Do

Integration cannot replace common sense. Until scientific knowledge and methods eliminate disease and other swine problems, an increase in size of business unit may lead to more problems than we now have. For example, a man who has been accustomed to raising pigs from 5 sows and does a poor job can be expected to do an even poorer job, in most cases, when he increases his operation to 40 or 50 sows. At the present time, this seems to be the great problem in many so-called "integrated" livestock programs. Keep in mind that diseased and sick pigs are of little value regardless of whether they are produced under integrated conditions. If one looks around the country and sees some of the empty farrowing houses and feeding floors brought about by poor sanitation and management, it becomes apparent that one man skilled in livestock management and sanitation practices is worth all the so-called "integrators" you could stack in a roundhouse.

Practicing veterinarians are the individuals upon whom a sound livestock industry depends. With disease control and good management, farmers can make some money from hogs, regardless of market conditions. Without disease control, the livestock industry cannot make a profit even with high prices.

Unfortunately, the veterinary profession has not done a good enough job of selling itself to American farmers. There are areas where veterinary "quacks," pseudo-educators, and pharmaceutical salesmen still are called upon to practice veterinary medicine. In many cases where there has been difficulty with disease control, it is not the result of competent advice by a veterinarian. It is usually the result of advice and action of a few untrained people who suggest such things as penicillin, streptomycin, tetracyclines, worm medicine, and what have you. Most of such advice given by this conglomeration of salesmen and self-styled veterinarians may be well intended, but it usually will end up causing heartache and economic loss to those who accept it. And let us not deceive ourselves into thinking that only nongraduate, nonlicensed people can be "quacks." A quack can also be a graduate veterinarian who through negligence and indifference does not do as good a job as he is capable of doing. If a veterinarian crawls through the fence with a needle in his hand and starts shooting without opening his eyes and making an honest attempt at diagnosis, he is, for that particular moment, a quack. Fortunately there are few veterinarians like this.

. . . let us not deceive ourselves into thinking that only nongraduate, nonlicensed people can be quacks.

The ease of procuring modern day drugs has been a boon to the veterinary profession, yet it also has its drawbacks. If a veterinarian makes a sloppy diagnosis and

tries to cover it by overzealous use of "miracle" drugs, it encourages sloppiness on the part of producers. It leads producers to put too much faith in hypodermic needles and syringes and not enough in common cleanliness and disease prevention. While veterinarians hold the key to efficient livestock production, a key which may be profitable, veterinarians also incur the obligation of showing the way and leading the fight in disease prevention. The time will come when livestock producers will keep veterinarians on a consulting basis—when financial livelihood is directly related to the number of disease problems veterinarians keep them out of, rather than the number of problems veterinarians get them out of after disease has struck.

Veterinarians are gradually building up support and are being recognized as an important part of the animal industry team. But they need, first of all, to give more attention to selling themselves to the people on the proper use of the local veterinarian and, second, to educate livestock producers in the use of disease prevention and sanitation programs.

Epizootiology, the Basis for Rational

Disease Control Programs

R. P. HANSON, PH.D.

IN THE GAME of caroms, a wooden ring is shot by a snap of the finger at other rings in the center of the board. The idea is to bounce one or more of the target rings into one of the four corner pockets. One of the group of target rings bunched in the center is easily hit, and the impact of the hit is passed on to adjacent rings in the group, often sending one or more into the pocket. After the board has been almost cleared, it becomes more difficult to score a hit on a target. Caroms illustrates two principles of epizootiology: (1) A hit or a transmission is accomplished by contact of one individual with another, the infected with the susceptible; (2) when the susceptible individuals are scattered, the chances of a hit or transmission are less.

Presented before the Wisconsin Conference on Tuberculosis Eradication, University of Wisconsin, Madison, Jan. 5-8, 1959.

Dr. Hanson is Professor of Bacteriology and Veterinary Science, University of Wisconsin.

Disease Transmission

Disease transmission, however, is much more complicated than is the carom game, because some of the "players" may be invisible. It is easy enough to follow the transmission of a disease like fowl plague which passes directly from one chicken to another. Live birds are susceptible and, when infected, develop signs of the disease and die.

It was less easy and required many years to unravel the more complicated story of yellow fever.[25] In the early days of the last century, 4,000 people (1 in 10) died in Philadelphia from "yellow jack." Yet the best medical men of that day said that quarantine for the disease was senseless, as the cause was a mist rising from coffee that was rotting at the Arch Street wharf. There are two reasons why they believed this: (1) People who nursed the sick at a hospital in the suburbs did not become infected, and (2) people who lived in the main part of the city near the wharf district, although they hid in their homes away from the sick and dying, became infected. Direct contact between diseased and susceptible individuals did not seem to be required. Now we know that the transmitting contact between a yellow fever patient and a susceptible individual is not by sneezing, touch, ingestion, or soiled clothing, but by bites of a mosquito that can pass through shuttered windows and doors. This mosquito bred in the Philadelphia of that day and was not present in the better drained and healthier suburbs.

The chain of transmission, even in apparent infection, can be diverse. The infective agent may be passed through the air from the nasal passages of one individual to the nasal passages of another by a sneeze or even in the eddy of an expired breath. This was first shown ten years ago when the virus of Newcastle disease was captured from the air of a California chicken house in which there were diseased chickens.[4] The virus particles, trapped by passing air through broth, reproduced the disease when inoculated into chickens. In Wisconsin a few years ago, a wind tunnel was set up[19] and a Newcastle disease-infected chicken was placed near the entrance. Susceptible chickens were placed at intervals along the tunnel. In only a few hours, virus from the diseased bird infected the susceptible chickens. Transmissible gastroenteritis of swine, distemper of dogs, and many other livestock diseases are also transmitted this way.

Some disease organisms gain entrance with the feed or water as droppings or scurf. A few years ago, a Wisconsin farmer selling cattle at the Milwaukee market was amazed to find that practically every animal in his herd was rejected for "measly" beef, because the flesh was peppered with the Cysticercus, the bovine tapeworm. These animals could have become infected with the larvae of this tapeworm only if they had ingested human feces. Subsequent investigation revealed that migrant workers at a nearby canning factory were defecating in the farmer's pasture.

The third means of transmission is from an infected individual to the susceptible one through an abrasion, such as a bite. Almost a year ago, a Wisconsin farmer lost 8 cows from a fatal infection that involved the central nervous system. The disease was diagnosed as rabies, and the farmer remembered that three weeks prior to the appearance of the disease a skunk had been observed in the barn and had presumably bitten the cattle before he escaped.

When the chain of infection involves individuals with inapparent disease—

Living Brucella in Butchered Sheep

Pathogenic *Brucella melitensis* were recovered from the tissues of 49 of 359 sheep from four flocks in which a high percentage of animals showed an allergic reaction on official testing. The organism was isolated from the uterus, spleen, liver and, in one case, from the neck muscles. This indicates a danger for persons handling the carcasses during and after slaughter. It also indicates that it might be well to destroy nonreacting as well as reacting sheep in heavily infected flocks.—*M. Lerche and H. J. Entel in Berl. u. Münch. tierärztl. Wchnschr. (Aug. 15, 1959): 319.*

animals that shed the infective agent without showing signs of disease—the chain of infection becomes difficult to follow. There are several types of inapparent carriers. Animals in the incubative stages of a disease, such as chickens having Newcastle disease, will shed virus for a full 24 hours before any signs of disease become apparent. Convalescent carriers, such as chickens infected with *Salmonella pullorum*, or animals with Brucella or Leptospira, will shed these organisms after they have apparently recovered from the disease.

Incubative and convalescent carriers are sometimes difficult to differentiate from "true carriers"—animals that never reveal by signs of infection that they have become infected. A cow may shed the Q fever organism in uterine discharges and in the milk and give no evidence of being infected.[24] It is probable that such an animal sheds Rickettsia for a relatively short time.

There are other infections in which the silent carrier continues to maintain or shed the infective organism for long periods. Deer have been shown in California to be carriers of anaplasmosis.[2] There is no sign of infection in these animals. The organism has not been demonstrated in blood smears and complement-fixing antibodies have not been found. Nevertheless, blood taken from deer is infectious for cattle by inoculation.

Another example of a long-term carrier is a mouse from whose feces lymphocytic choriomeningitis virus may be isolated.[10] This virus disease can produce fatal infection in man, dogs, and mice. In certain colonies of mice, the disease becomes established as an inapparent infection. No antibodies to the virus develop in carriers. The normal span of life is lived, the virus is transmitted from the mother to her offspring before birth, and each successive generation continues to shed the virus in its feces. This latent or carrier state is dependent upon an adaptation between the host and its parasite and not a genetic change in either. The virus remains fully pathogenic for susceptible mice of other colonies. The inapparent carrier is one means of undercover or invisible transmission of disease, since contact of apparently infected individuals with the susceptible is never seen.

Another important means of undercover transmission is through vectors, usually an arthropod, although in recent years helminths and other invertebrates have been incriminated. Some vectors transmit mechanically; the transmitting insect behaves like a flying needle, first contaminating its biting parts with the infective agent and then passing the agent on when it bites another host. Fowlpox of chickens is transmitted in this way. Mosquitoes, in biting through the pox lesions, contaminate the biting parts. The virus is then injected into the next chicken.

The inapparent carrier is one means of undercover or invisible transmission of disease, since contact of apparently infected individuals with the susceptible is never seen.

Mechanical transmission is effective as a means of spreading disease. The story of myxomatosis is an excellent illustration. A misguided hunting enthusiast years ago introduced the European rabbit into Australia where it multiplied so prolifically and spread so rapidly that it became one of the most serious agricultural pests in history. The sheep range in Australia was being destroyed by the rabbit. Poisoning, hunting, and other means of reducing the rabbit population were tried. Then, less than ten years ago, the Australian government introduced a disease of rabbits, myxomatosis, that had been discovered years earlier in South America.[6] In the laboratory, myxomatosis had been shown to cause almost 100 per cent fatality in rabbits of the European species. The first attempt at introduction failed, because the Australians believed that the virus was spread by contact and chose the wrong place and season. Recognizing later that myxomatosis virus was spread by mosquitoes flying from one rabbit warren to another and by fleas within the warren, the Australian virologists timed the introduction of the disease to coincide with the seasonal peak in mosquito population. It was successful; the disease spread rapidly through the range country of Australia, destroying tremendous numbers of rabbits.

A recent visitor to the United States from Australia reported that the Aus-

tralian parliament now believes that the introduction of myxomatosis has meant a $19 million increase in the annual income of sheepmen, because of both the increase and the improvement in the ranges, which followed the destruction of rabbits. This is an instance in which a disease was used to improve agricultural production by reduction of an undesirable species. The significant thing to us is the effective distribution of the virus from rabbit warren to rabbit warren over 1,000 miles of range land by a biting arthropod. Without the agency of the mosquito in the first introduction, the disease failed to spread and disappeared.

A biological vector cannot transmit virus immediately after ingesting it as can mechanical vectors.

A vector of disease need not always pierce the skin. An epizootic in which a nonbiting vector played a role occurred in northeastern Wisconsin.[23] Botulism of pheasants is caused by a toxin that is produced by an anaerobic bacterial organism that often multiplies in the carcasses of dead animals. Pheasants may become poisoned by pecking and eating flesh from dead carcasses or by eating maggots of flies that have bred in these carcasses. The maggots and flies are not harmed by the poison, but they may contain enough of it within their bodies to cause disease and death in birds that ingest a few larvae or adults. Movement of maggots and flies from pen to pen apparently spread the disease in the Wisconsin epizootic.

Biological vectors of disease have been recognized since the days of Theobold Smith. These are usually arthropods in which large parasites or the smallest viruses are able to multiply.[5] A biological vector cannot transmit virus immediately after ingesting it, as can mechanical vectors. During this incubation period, the parasitic agent multiplies and moves from the gut into the salivary glands. After hours or days, the vector is capable of injecting virus with saliva as it feeds on new hosts. Yellow fever of man and malaria of birds are transmitted in this way by mosquitoes. Rocky Mountain spotted fever and other rickettsial disease are transmitted in a similar manner by ticks or mites.

The transmitting agent need not always be alive. Inanimate instruments of transmission are called fomites. They include hypodermic needles, boots and feed sacks, automobiles, and shipping crates.[21] Anthrax, a spore-forming agent, a cause of a fatal infection in several types of livestock, has been effectively transmitted by fomites, as in a shipment of contaminated shaving brushes made from bristles of infected pigs.[22] The spores were not destroyed in the process of manufacture of the brushes and lay in wait upon the hairs until an abrasion on the face of a man permitted entry. Several fatal infections resulted. The U.S. Department of Agriculture has devoted considerable time to the study of anthrax in the lower delta of the Mississippi.[20] In that area, the spores are spread by floods over the land in spring. Vultures and other scavengers can distribute anthrax to areas unreached by floods as the spores pass unharmed through the digestive tracts of these scavengers and are dropped in their feces over the countryside.

In 1952, anthrax appeared in many parts of Wisconsin and other states where the disease had never been previously reported.[26] Although the source of infection has not been entirely proved, the evidence incriminates a bone meal which was fed these animals. The bone meal was imported to the United States from Africa and other places in the eastern hemisphere where anthrax is enzootic. It included the bones of domestic animals, elephants, and even man that either had been ground and prepared without steam sterilization or shipped in vessels in which unsterilized bone meal was also shipped. Cattle, swine, and horses died in Wisconsin. The infection chain did not end there. One fur farmer buying horses that had died of anthrax and suspecting that perhaps they had had the disease, fed the meat first to foxes which were worth little to him and which, unknown to him, were also resistant to the disease, and then to his expensive mink which are, unfortunately, susceptible to anthrax. The foxes survived; the mink died. Since it was winter and the

pelts were prime, the farmer salvaged the pelts, although they were contaminated with anthrax spores. Attempts to decontaminate them by sterilization with ethylene oxide gas failed and the pelts had to be destroyed.

Another farmer was foiled in his attempt to perpetuate a cycle that could have included man. When his swine became sick with anthrax, he salvaged them by butchering and then pickling the pork. He stored the pork in his basement. Only by court order was he compelled to destroy the meat. He intended to feed it to his family.

Epizootiology and Disease Control

Epizootiology involves apparent disease, inapparent disease, carriers, vectors, and fomites. It involves such other important factors as spatial relationships and the relative abundance of hosts and vectors. Newcastle disease virus transmitted by aerosol does not get very far when there are only a few chickens on a farm and the farms are widely separated. At present, an intensive poultry industry with a thousand birds to a house, 10 to 100,000 birds to a farm, invites a disease transmitted by aerosol to spread with lightning rapidity.

Changes in animal husbandry that have come about with a need for greater farming efficiency have increased the number of animals of given species of livestock on a farm whether it be chickens, swine, or cattle. More and more, animals are being concentrated into a given area. At the same time, we have been breeding animals for feed conversion efficiency, for greater egg production or milk production, and with little or no attention to factors that make for disease resistance. Consequently, we will do well to keep abreast of new problems in spite of our increased attention to disease research and our improved methods of diagnosis and improved means of controlling disease.

Some of the principles and facts on which a disease control program must be based have been discussed. There are many unsolved problems. We know little about the wildlife reservoirs of most of the diseases of livestock. The large liver fluke, *Fastioloides magna*, causes an inapparent disease in deer and a fatal infection of sheep.[16] On one northern Wisconsin farm on which this disease had caused considerable loss, the daily movement of the sheep flock and of a herd of deer was studied. They crossed regularly at one point in the farm woodlot, a marshy spot where there was an abundance of snails, which serve as an intermediate host for the fluke. There are 850 different species and subspecies of snails in Wisconsin. Which snail is the intermediate host for this parasite has not been determined.

Eastern equine encephalomyelitis and several other insect-borne viruses are present in Wisconsin.[12] On one Wisconsin farm, 32 species of mosquitoes, 11 biting midges, 21 horseflies, and 19 deer flies were identified.[17] One or more of these 83 species of insects are vectors of livestock diseases; which ones we do not know.

How do some of the disease agents survive in the period between epizootics of the disease? It has been proposed that swine influenza and hog cholera viruses can persist in the swine lungworm that spends its larval stages as a parasite of earthworms.[18] Perhaps other diseases survive between outbreaks in livestock in a similar manner.

New diseases of livestock are frequently reported. From where do they come? Newcastle disease virus spread from the Dutch East Indies to most of the nations of the world in a period of 15 years.[2] Have these diseases existed previously in the United States in some unrecognized form? It has been suggested that Newcastle disease was present in the United States long before it was recognized as a new disease.[1] Did they hide in another species of livestock, in man, or in wild animals? This appears to be the story of vesicular stomatitis.[12] Is the dis-

Epizootiology is not a problem for the research worker alone. Alert practitioners, making direct observations, may reveal the logical experimental approach to some of the problems.

ease a result of a new association between the host and its parasite? The last may well be true. Some poultry pathologists believe that this is the origin of chronic respiratory disease.

There are at least four sleeping plagues in the upper Mississippi Valley. Antibodies for eastern equine encephalomyelitis have been found in the wild birds in all parts of Wisconsin. Virus has been isolated from apparently normal pheasants and from a few diseased horses. The prevalence of antibodies for the virus in birds seems to be about the same in Wisconsin as it is in Massachusetts where fatal cases in man and horses are reported every few years. Why have there been no reports of eastern equine encephalomyelitis in man in Wisconsin and only a few reports of the disease in horses?

Within the past few years, we have learned that Q fever is a relatively common infection of dairy cattle in southern Wisconsin, northern Illinois, and eastern Iowa.[13,15] In Italy, Australia, and California this has been a common disease of man. The disease itself is not apparent in Wisconsin cattle and, with the exception of one clinical case, apparent disease has not been reported in man.

A short time ago there were accounts in the newspapers of severe illnesses and deaths in Texas and in Oregon of people who handled turkeys affected with psittacosis.[9,11] In both of those areas turkeys also died of the disease. On the basis of serologic findings, we know that psittacosis is prevalent in both turkeys and pigeons in all parts of Wisconsin and in much of Minnesota.[8] With the exception of a dozen mild human cases reported from one locality, the disease has not been recognized in man in Wisconsin. In turkeys, the disease has been inapparent.

Serologic studies have revealed that leptospirosis is relatively widespread as a parasite of cattle and deer in all of the North Central States.[7] Relatively few problem herds have been reported among Wisconsin cattle, and human infections have not been clearly traced to cattle. Leptospirosis has been shown to be a serious disease of cattle in some areas of the world and is a serious and sometimes a fatal infection of man in the Middle East.

Why is it that these diseases are not more serious to livestock and to man in the Midwest? Most people would agree that this question deserves an answer.

Conclusion

Epizootiology is not a problem for the research man alone. The alert veterinarian has an opportunity to make direct observations that can suggest an experimental attack on some of these riddles. Observations which he transmits to investigators and field materials which he refers to laboratories may provide the necessary source for a new and profitable lead.

The observer of epizootiologic phenomenon is encouraged to communicate with the man who is planning the next move in scientific control of animal disease.

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Coccidioidomycosis, from Man to Dog

A 4-year-old Golden Retriever bitch, that had wintered in Arizona and returned to New Hampshire for the summer, was admitted to a veterinary hospital in July with lameness in the right hind leg and a discharging sinus on the medial aspect of the lower tibial region. The affected leg was quite lame, the skin of the medial aspect of the right thigh was erythematous, and yellow serous fluid dropped from a 3-cm. circular opening at the distal end of a marked tibial swelling.

Lameness had begun in May, accompanied by a slight fever, and was followed by an intestinal upset in early June. In late June, a cutaneous lesion appeared on the medial surface of the tibial region and was given local treatment. The lesion and the lameness worsened, and a bony swelling and an open discharging sinus at the tibial lesion appeared.

Radiographs showed a bony lesion extending from the lower 1/8 of the shaft to a point ending at the upper third of the shaft. The swelling appeared to be subperiosteal and smooth except for a rough area at the proximal end. The medullary cavity showed destruction in the center of the lesion and proliferation at either end.

The lungs showed very slight areas of density, detected only in retrospect.

Tentative diagnosis of bone infection and speculative diagnosis of coccidioidomycosis were made. The leukocyte count was 19,950 and hemoglobin 12.2 Gm./100 cc. Discharging pus was examined for *Coccidioides immitis*.

After initial diagnostic studies were completed, it was learned that the dog's master had contracted and recovered from coccidioidomycosis in Arizona. The dog was euthanatized.—*Veterinarian (Mass. V.M.A.) 4, (1959): 22.*

Sterile Disposable Swabs

for Bacteriological Diagnosis

M. B. TEIGLAND, D.V.M.
V. R. SAURINO, PH. D.

THE MEDICAL laboratory can provide a vital auxiliary service for the diagnosis and prognosis of a vast majority of the diseases seen by clinicians. The reliability of the laboratory report, however, is completely dependent on how efficiently a specimen is collected and submitted for analysis. Contamination and deterioration of the specimen can nullify any efforts toward its analysis.

The disposable equipment described herein is designed to facilitate the collection, and to minimize the contamination, of specimens for bacteriological analysis. Three types of sterile, disposable, plastic-tubed swabs (fig. 1,2) are described.

Large Animal Nasopharyngeal Swab

This swab consists of 3 telescoped plastic tubes contained in a thin plastic bag. After removal from the bag, the entire assembly of tubes is passed through the nostril in the same manner as a stomach tube. When the back of the throat is contacted, the middle tube is gently pushed forward in order to open the protective flap on the outer tube. The innermost third tube, containing the attached swab, is pushed, finally, against the mucous membrane. After collecting the specimen in this fashion, the innermost third tube can be withdrawn into the middle tube and the entire apparatus of tubes can be removed from the nostril.

Dr. Teigland is associate research professor, and Dr. Saurino is associate professor, Department of Microbiology, School of Medicine, University of Miami, Coral Gables, Fla.

All swabs described herein are manufactured by Dade Reagents, Inc., Miami, Fla., and are distributed by Walter May, 743 N.W. 41st St., Miami.

Large Animal Uterine Swab

This is an assembly of two sterile plastic tubes: a rigid outer tube sufficiently long to reach the cervix of a mare or cow and an inner swab-attached tube covered by a thin plastic bag. The entire apparatus, including the bag, is inserted into the vagina until contact is made against the cervix. The protective bag is then removed by holding the rigid tube firmly in one hand and steadily pulling back on the bag with the other hand until the bag tears away from the tubes. The rigid outer tube is inserted into the uterus, and the inner swab tube is pushed out to contact the membranes and then withdrawn in the protection of the rigid outer tube; finally, the entire apparatus is removed. A scored ring, 6 inches from the anterior end of the rigid outer tube, allows the operator to easily snap off the tip of this tube (fig. 3) and thereby aid in minimizing possible contaminants from the process of withdrawal.

Small Animal Uterine, Throat, or Body Cavity Swab

This is a semirigid plastic assembly of 2 tubes: outer protective and inner swab-containing. Bacteriologic specimens from membranes or cavities can easily be obtained by the methods described above.

Culturing Procedure with the Disposable Swabs

The tip ends (about 4 to 6 in.) of the swab-containing tubes should be transferred to a sterile broth medium as soon as possible after the specimen is taken. This process simply involves the cutting

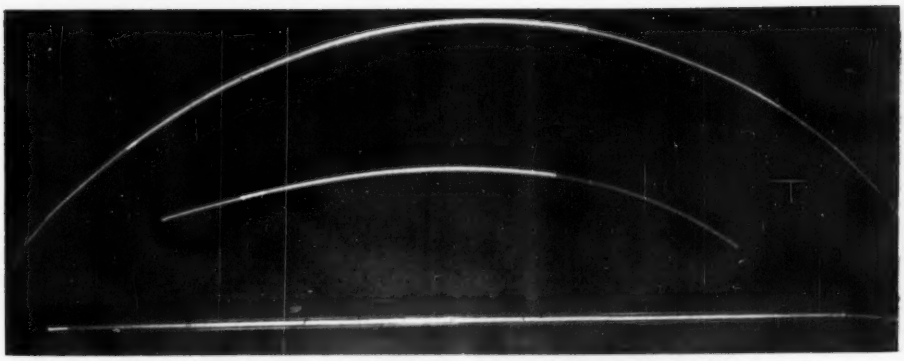


Fig. 1—(Top)—Large animal nasopharyngeal swab; (center)—small animal uterine or pharyngeal swab; (bottom)—large animal uterine swab.

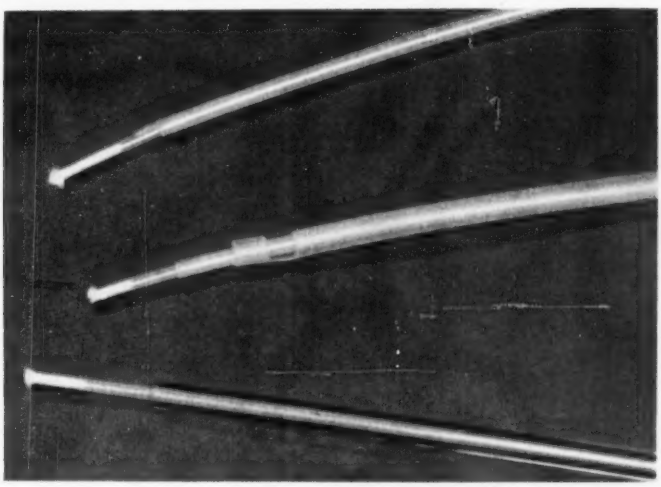
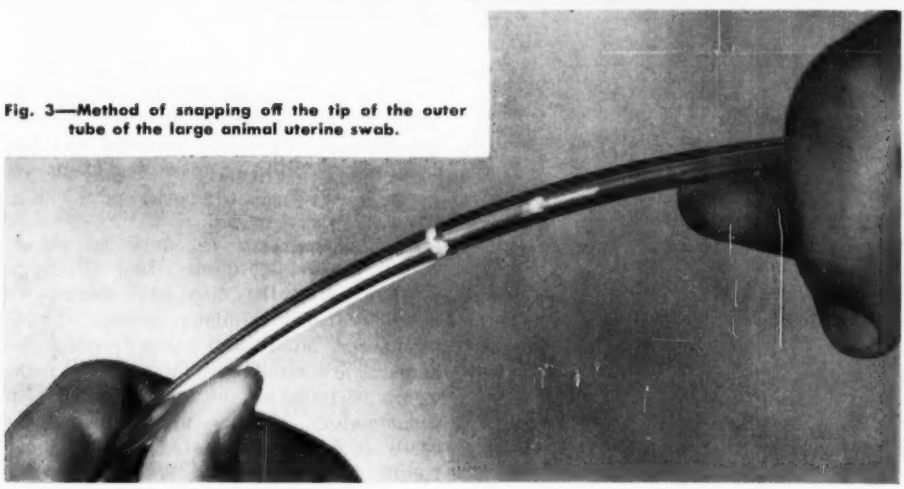


Fig. 2—Swabs exposed: (top)—small animal uterine or pharyngeal swab; (center)—large animal nasopharyngeal swab; (bottom)—large animal uterine swab.

Fig. 3—Method of snapping off the tip of the outer tube of the large animal uterine swab.



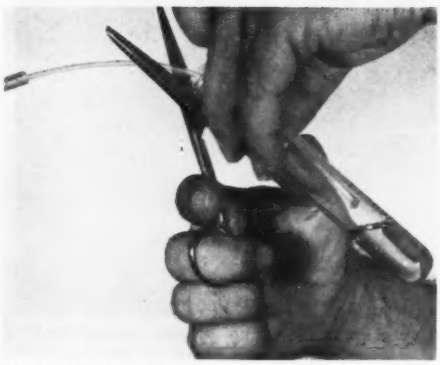


Fig. 4—Cutting off swab and introducing in tube of medium.

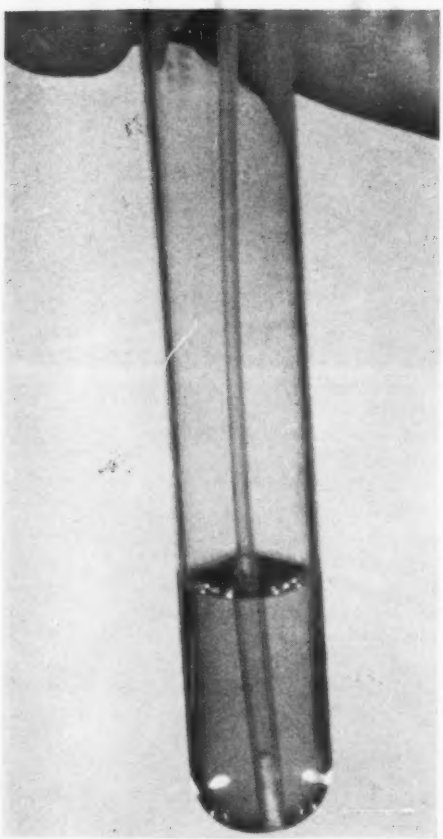


Fig. 5—Swab in tube.

off, with sterile scissors (fig. 4) of about 4 inches of the immediately adjacent protective tube. (In the field, scissors may be sterilized by dipping into 70% alcohol and igniting.) The swab is pushed directly into the mouth of a culture tube and a few inches of this swab tube is cut off aseptically (fig. 5). Screw-capped culture tubes containing about 1 inch of sterile Todd-Hewitt or brain heart infusion broth are used. The swab is allowed to drop into the medium and the tube is recapped tightly. This makes a convenient transporting device and prevents destruction of organisms by drying.

Procedure at Laboratory

On arrival at the laboratory, the swab is aseptically withdrawn from the test tube and touched onto the margin of one blood agar plate, then streaked over the entire surface of a second one. The swab is then discarded but the carrying broth medium is kept in the refrigerator for any studies that may seem desirable later. The drop on the first plate is streaked with a sterile wire loop. This plate is used for isolation and identification of organisms. The second plate is used for antibiotic sensitivity testing by placing standard antibiotic discs in circular arrangement on the culture surface.

Usually the plates are read within 24 hours. The colonies on the isolation plate can be further processed, streaked, and stained for identification. The sensitivity antibiotic disc plate will indicate the antibiotics to which the organisms are sensitive. A report to the veterinarian can be submitted in 24 hours and could include the type of organisms involved and the drug susceptibility of these organisms.

Contaminants Minimal

When using the disposable swabs, we have obtained a minimum amount of contaminants. Molds and spore-forming organisms are rarely encountered. The pathogenic bacterial organisms, associated with inflammatory mucous membranes, were easily and clearly isolated by the above technique.

Leukemia

and Related Neoplasms

in the Cat

II. Malignancies Other than Lymphoid

Jean HOLZWORTH, D.V.M.

LEUKEMIAS AND TUMORS of lymphoid and reticulum-cell type were described in a previous paper (JOURNAL, Jan. 15, 1960: pages 47-69.)

Myeloid Leukemia

Malignancies involving cells of the myeloid series are far less numerous in the cat than those of the lymphoid type, there being only a few well-documented reports of their occurrence.[45-52]

Nine cases of myeloid leukemia were encountered in cats at the Angell Memorial Hospital. The majority were males and, in color, tabbies, black, or black and white. There was 1 Siamese. Two were less than a year old, 2 were young adults, the rest were middle-aged, ranging from 3 to 10 years. One cat with hematologic findings characteristic of a chronic myelocytic leukemia had a long record of infected bite abscesses and upper respiratory infection. The rest were observed to be ill only for periods ranging from a few days to three weeks. Clinical signs were poor spirits and appetite, decreased activity, weakness of the limbs, thirst, diarrhea, dyspnea, and vomiting. One cat ate sand. Fever ranging from moderate to pronounced was present in half. Enlargement of the liver and spleen was recognized in the majority. In some the superficial nodes were moderately enlarged; in 1 only one prescapular node was grossly involved. One cat had a hemorrhagic tendency, bleeding profusely from the nose and gums and at sites of venepuncture. Mild jaundice was observed in several.

In 3 cats, the total white blood cell count was below normal, but in the others there was a significant elevation, the highest being 65,000. Anemia was severe in all cases, hemoglobin ranging from about 2.0 Gm./100 ml. in one of the most acute to 5.5 in a chronic form.

In general, myelocytes predominated in the blood—huge cells with large, round, often eccentric, reddish purple nuclei and rather deep blue cytoplasm with reddish granules (fig. 27). Although it is often hard to distinguish myelocytes from other mononuclear cells with azurophilic granulation, such as monocytes or large lymphocytes, there were often sufficiently numerous myeloid cells of maturer form so that the immature ones could be recognized by the company they kept (fig. 27). In some cases, the differentiation was made by means of a peroxidase stain, peroxidase-positive granules occurring in the cytoplasm of cells of the neutrophilic series of the stage of myelocyte and older (and in smaller number in monocytes) but not in lymphocytes. In the more acute cases myeloblasts and mitotic forms were frequent (fig. 30).

Platelets were sometimes increased in number.

In several cats there were large numbers of nucleated red blood cells, some very primitive. In 1, these so far outnumbered all other nucleated cells as to suggest at first sight erythroleukemia (fig. 31).

The most consistent and prominent organic change was enlargement of the spleen, which in 1 cat attained a length of over 25 cm. and lay across the abdominal floor with ends curved upward. In another cat, rupture of the greatly enlarged spleen was the immediate cause of death. In contrast with its appearance in lymphoid malignancies, the spleen in the myeloid leukemias tended to be of a rather homo-

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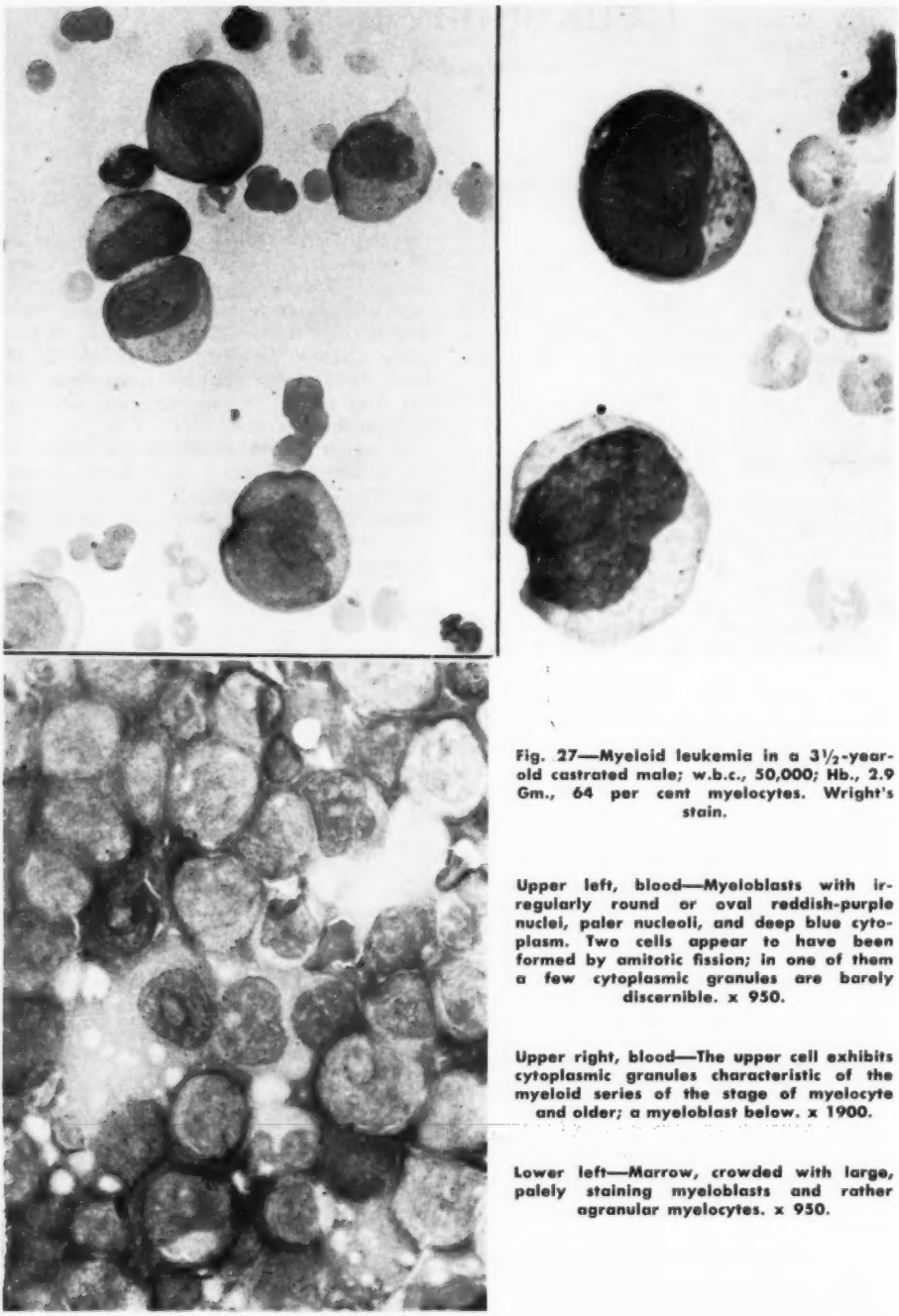

Fig. 27—Myeloid leukemia in a 3½-year-old castrated male; w.b.c., 50,000; Hb., 2.9 Gm., 64 per cent myelocytes. Wright's stain.

Upper left, blood—Myeloblasts with irregularly round or oval reddish-purple nuclei, paler nucleoli, and deep blue cytoplasm. Two cells appear to have been formed by amitotic fission; in one of them a few cytoplasmic granules are barely discernible. x 950.

Upper right, blood—The upper cell exhibits cytoplasmic granules characteristic of the myeloid series of the stage of myelocyte and older; a myeloblast below. x 1900.

Lower left—Marrow, crowded with large, palely staining myeloblasts and rather agranular myelocytes. x 950.

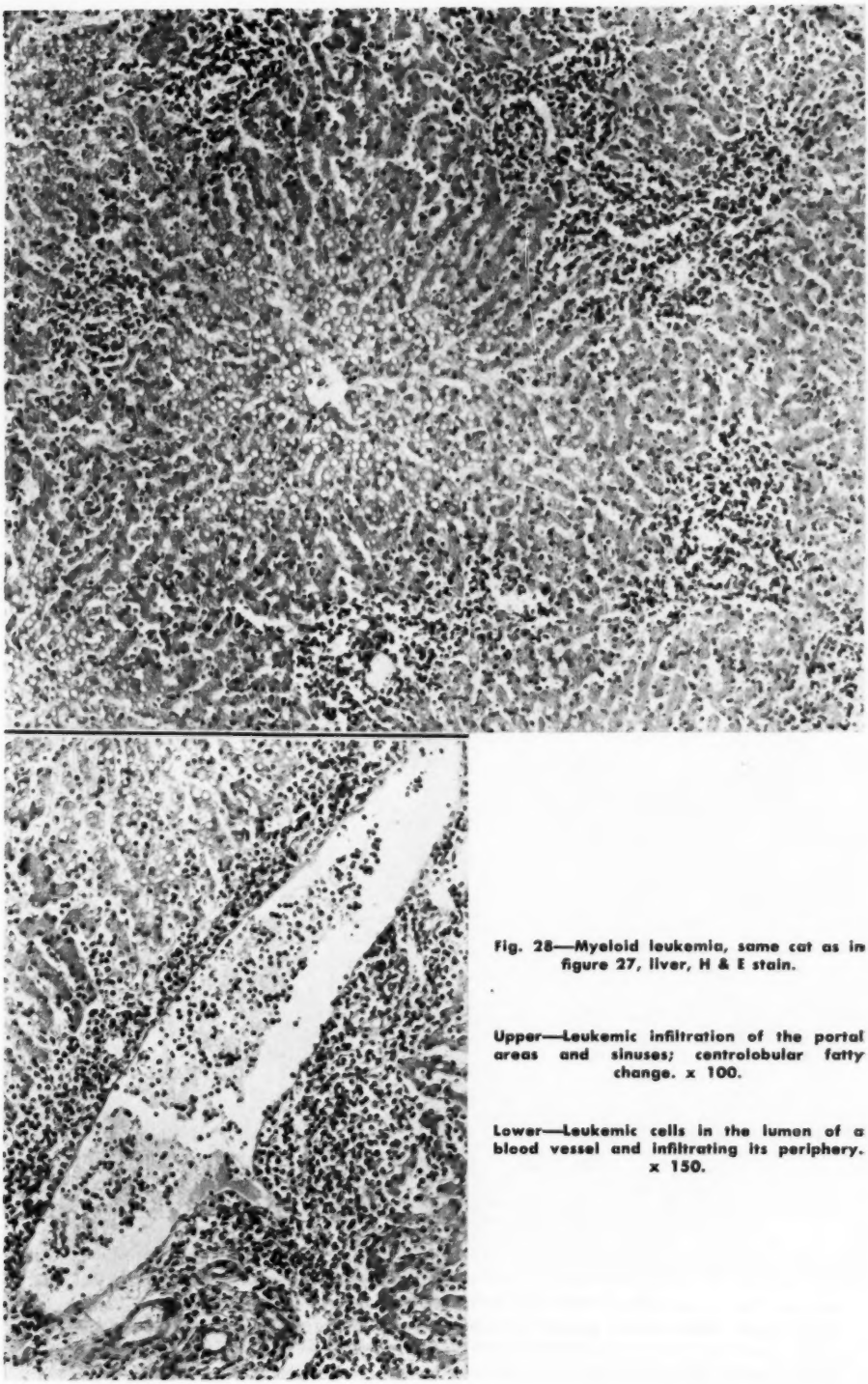

Fig. 28—Myeloid leukemia, same cat as in figure 27, liver, H & E stain.

Upper—Leukemic infiltration of the portal areas and sinuses; centrolobular fatty change. x 100.

Lower—Leukemic cells in the lumen of a blood vessel and infiltrating its periphery. x 150.

geneous blackish purple, with little or no trace of lymphoid follicles.

Microscopically there was usually atrophy or obliteration of the splenic follicles and diffuse infiltration of the red pulp by immature myeloid cells (fig. 29). In the more acute cases, myeloblasts and mitotic forms were numerous. Leukemic cells packed the marginal sinuses and crowded the lumina of vessels, invading their walls and accumulating beneath the intima. The splenic capsule and septa were also invaded. Megakaryocytes were often present in great numbers, and foci of red blood cell formation were sometimes conspicuous.

In most cats, the liver was enlarged in varying degrees (to 255 Gm. in 1), sometimes with a grayish mottling or foci. Irregular dark red infarcts were observed occasionally. The portal areas and sinuses were infiltrated with myeloid cells, and similar cells were often scattered diffusely through the parenchyma. Centrolobular atrophy and fatty change were observed in some cases (fig. 28). Megakaryocytes were sometimes numerous, and foci of hematopoiesis were often prominent.

The lymph nodes generally were much less prominently involved than in lymphoid malignancies, sometimes only a single node or group of nodes undergoing enlargement. Microscopically they exhibited reticular hyperplasia and infiltration by myeloid cells. The lymphoid tissue was reduced, sometimes almost to the point of obliteration (fig. 29). Megakaryocytes and foci of red blood cell formation were often seen.

The bone marrow in the myeloid leukemias gave an impression of tremendous cellularity, with intense diffuse proliferation of myeloid forms, among which myeloblasts and myelocytes predominated (fig. 27, 30). Mitotic forms were often numerous. In 1 cat, mature eosinophils were greatly increased in number. The hyperactivity of the marrow was often reflected

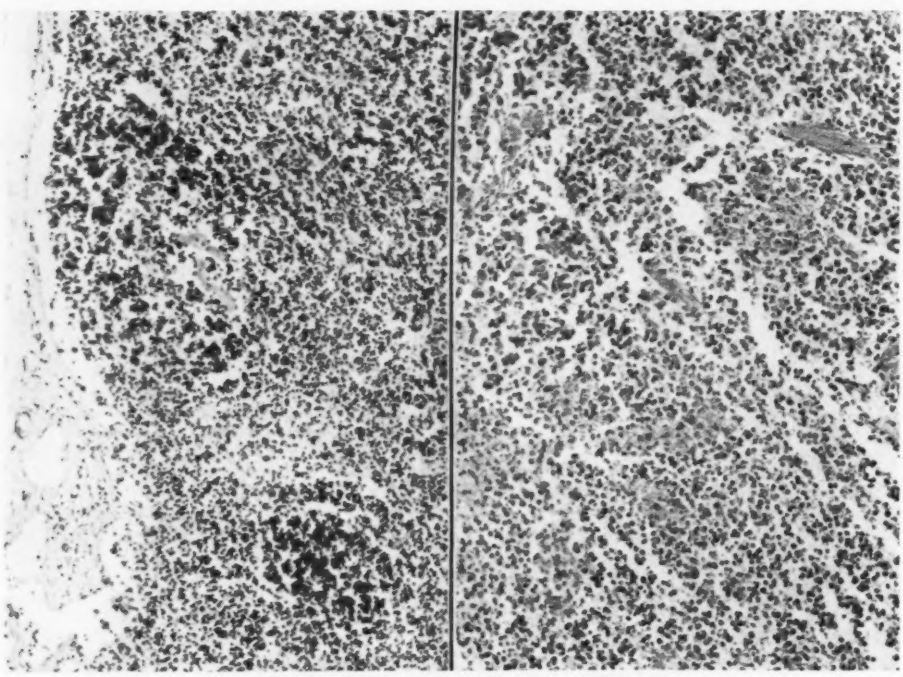

Fig. 29—Myeloid leukemia, same cat as in figure 27.

Left, lymph node—Severe general infiltration with myeloid cells and partial obliteration of lymphoid nodules. H & E stain; x 100.

Right, spleen—Blurring of splenic architecture by infiltrating myeloid cells. H & E stain; x 150.

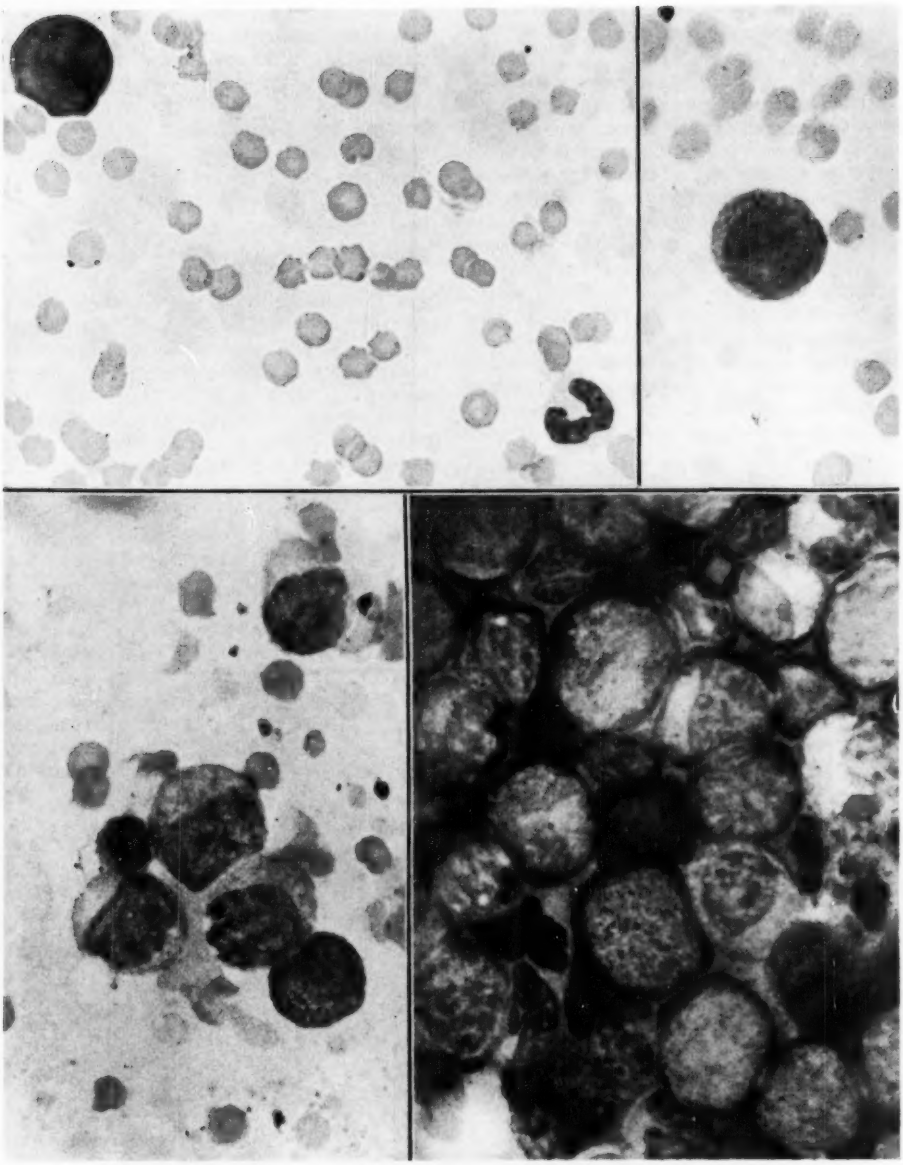

Fig. 30—Myeloid leukemia in an 11-year-old spayed female; w.b.c., 3,700; Hb., 3.5 Gm., Wright's stain.

Upper left—Myeloblast and band neutrophil. x 950.

Upper right—Myelocyte with numerous granules. x 1200.

Lower left—Liver impression: Myeloblasts and myelocytes with one nucleated red blood cell. x 1200.

Lower right—Marrow crowded with pale-staining myeloblasts, myelocytes, and band neutrophils. x 1200.

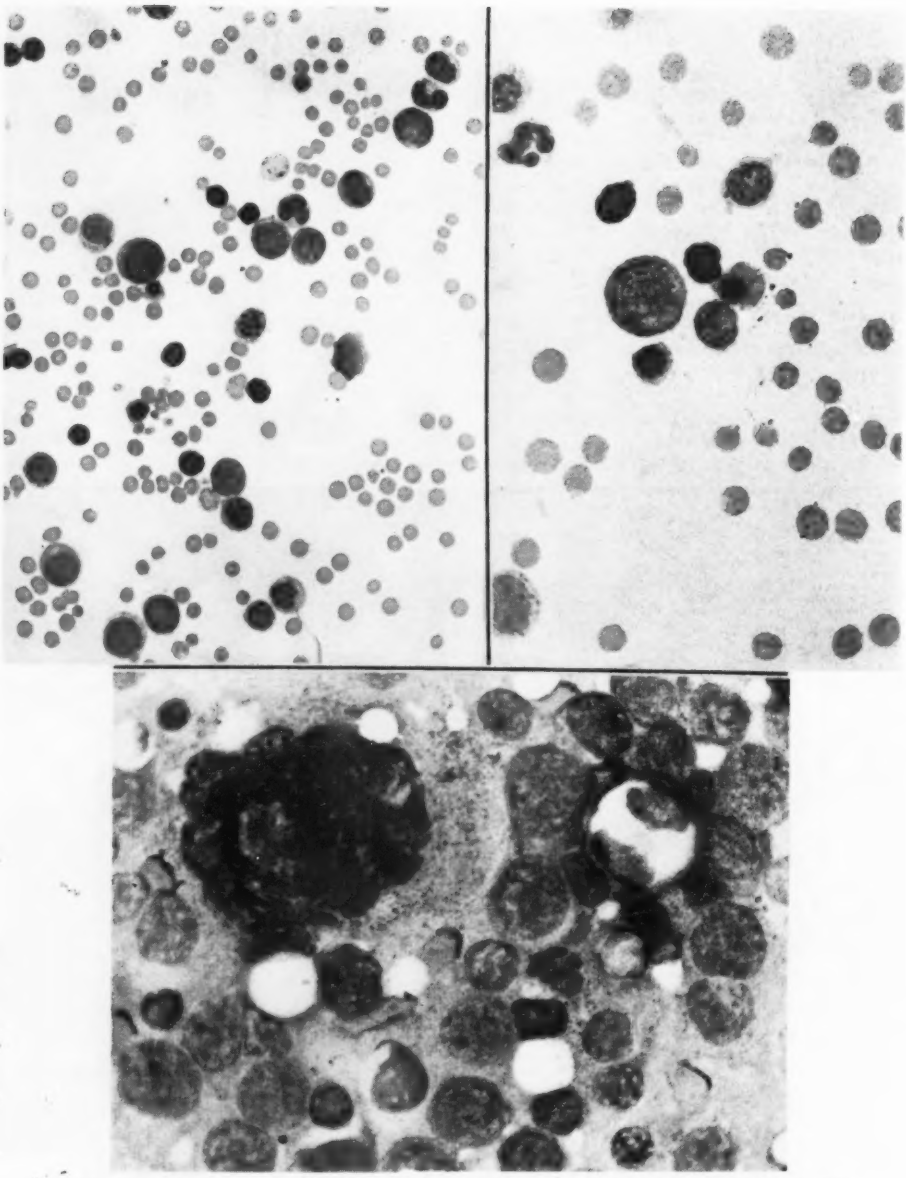

Fig. 31—Myeloid leukemia in a 2-year-old female; w.b.c., 60,000; Hb., 2.6 Gm. Wright's stain.

Upper left, blood—Myelocytes and metamyelocytes predominate, but an almost equal number of nucleated red blood cells is present. x 525.

Upper right—Two large and two smaller myelocytes with a scattering of cytoplasmic granules. x 950.

Lower, marrow—Myelocytes and myeloblasts predominate. There is a megakaryocyte (upper left) and a cell in mitosis (upper right). x 950.

in the presence of increased numbers of megakaryocytes and, although anemia was severe, there was frequently tremendous activity of the surviving erythroid elements (fig. 31).

Small foci of leukemic cells were sometimes observed microscopically in other organs, notably the kidney, heart, and lung.

Basophilic (Mast-Cell) and Eosinophilic Leukemias

Rare varieties of myeloid leukemia are those in which basophilic or eosinophilic granulocytes predominate. The occurrence of *basophilic myelocyte leukemia* in cats was mentioned, without details, by one writer,[54] and two others[56,58] each reported a case having in common the feature that neither cat was known to be ill but died suddenly, the 1 after an intraperitoneal injection and the other of rupture of the spleen. A recent study of mast-cell malignancies describes four additional cases of mast-cell leukemia, all in elderly castrated male cats.[55]

An instance of basophilic leukemia was encountered at the Angell Memorial Hospital in a cat from which an exceedingly large spleen was removed. Subsequent studies revealed total white blood cell counts ranging between 24,000 and 31,000/cmm. with 4 to 7 per cent basophils. Unlike the great majority of leukemias in cats, this was not accompanied by severe anemia.[57]

Myeloid leukemia of the *eosinophilic* variety appears to be even more of a rarity. The only instance known to the author was observed by a practitioner[59] whose diagnosis was confirmed by the clinical pathologist at his state's veterinary college.[60] The cat, an 8-year-old castrated male, had a large spleen and liver, a total white blood cell count of 150,000/cmm., and moderate anemia. He had lost energy and weight, vomited, and had diarrhea. The differential count was: segmented eosinophils 46 per cent, band eosinophils 22, eosinophilic metamyelocytes 3, eosinophilic myelocytes 2, neutrophils 15, lymphocytes 9, and monocytes 3. There were a few primitive or "blast" forms (fig. 32). One of several fecal examinations disclosed coccidial oocysts. The cat died two months after the condition was diagnosed, during an intravenous injection of a sulfonamide as therapy for the coccidial infection. Permission for a necropsy could not be obtained.

Monocytic Leukemia

Monocytic leukemia is a relatively uncommon dyscrasia in man and has been the subject of controversy owing to differences

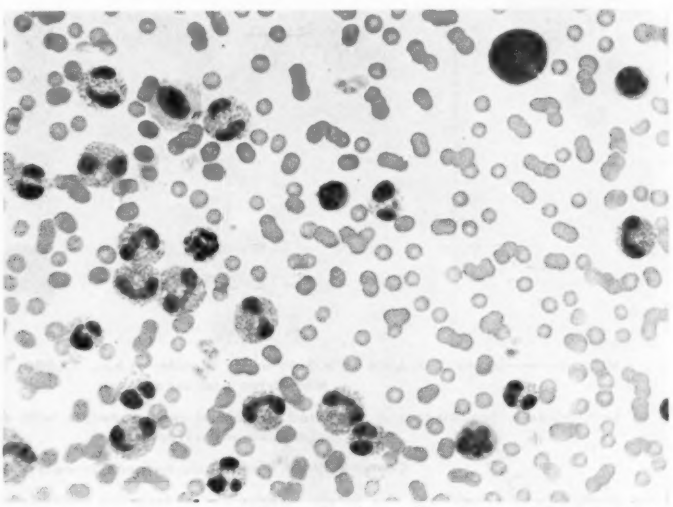

Fig. 32—Eosinophilic leukemia in an 8-year-old castrated male; w.b.c., 150,000; moderate anemia; 73 per cent eosinophils. Segmented and band eosinophils predominate; "blast" form, upper right. Wright's stain; x 400.

—Film lent for photographing, Courtesy of Dr. Oscar Schalm

of opinion as to the origin of the monocyte and the difficulty of demonstrating it in fixed tissues without the aid of special methods. Anatomically, this type of leukemia is usually characterized in man by less striking enlargement of nodes, liver, and spleen than in the other leukemias.

In 1 cat in the Angell Memorial group, monocytic leukemia was positively diagnosed antemortem by supravital blood studies.

A 3-year-old tricolored female had aborted several months before her final illness. Two days before admission she suddenly became depressed and inappetent, crying out and vomiting. Examination disclosed pallor, apathy, constipation, and a normal temperature. During six weeks of hospitalization, the cat would brighten and eat well after blood transfusions, but despite administration of hematopoietic agents the anemia remained refractory, the hemoglobin ranging persistently between 2 and 5 Gm./100 ml., although megaloblasts, normoblasts, and basophilic red blood cells were numerous. Rouleaux-formation was pronounced. Although the total white blood cell counts remained substantially within the normal range (8,000 to 17,000), about 45 per cent were large mononuclear cells, of which the majority were tentatively identified as monocytes by their blue-gray cytoplasm, azurophilic granulation, and indented or slightly lobulated nuclei with coarse linear chromatin. Some of this group were "blasts," with spherical leptochromatic nuclei and from one to several nucleoli. Their cytoplasm was but a small, dark blue rim (fig. 33). "Smudge" forms suggested increased fragility. Small numbers of peroxi-

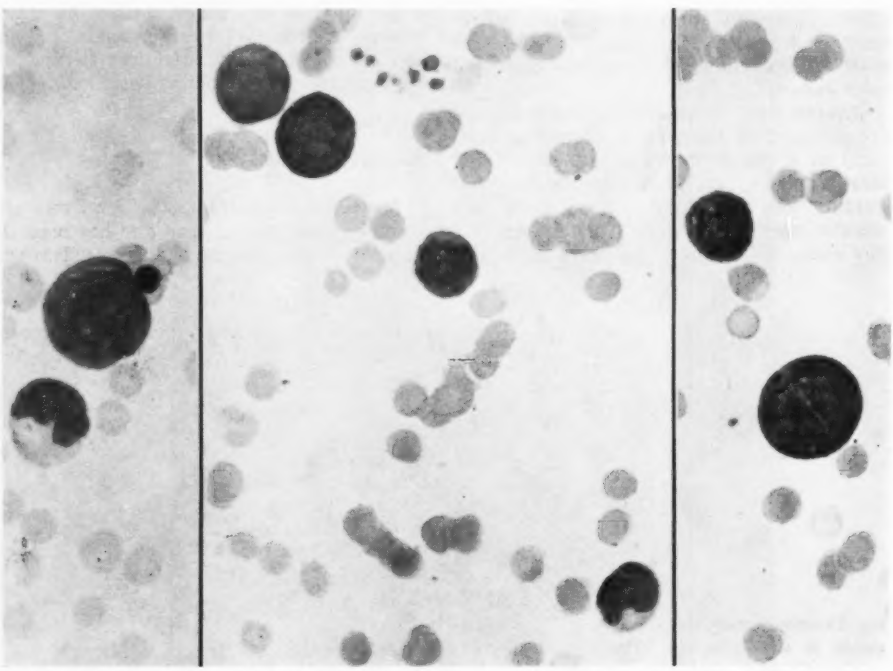

Fig. 33—Monocytic leukemia in a 3-year-old female; w.b.c., 12,000; Hb., 4 Gm.; 45 per cent monocytes and monoblasts.

Left, blood—Monoblast and nucleated red blood cell, monocyte with coarse lobulated nucleus and cloudy cytoplasm.

Center—Three monoblasts and one monocyte.

Right—Two monoblasts, the larger apparently undergoing amitotic division. Wright's stain; x 950.

dase granules were present in the maturer forms. Only by means of supravital studies, which disclosed typical movements and staining properties of monocytes, was it possible to distinguish these cells with certainty from myelocytes. The blood platelets were decreased.

At necropsy, the predominant cell in the marrow was found to be a "blast" with basophilic cytoplasm and a round leptochromatic nucleus with a variable number of nucleoli. These cells were mingled with an almost equal number of immature red blood cells (fig. 34). Granulocytes were rare and their development was arrested at the stage of "band" forms. Megakaryocytes were also rare.

Wright- and Giemsa-stained impressions of the lymph nodes, liver, and spleen disclosed large numbers of mononuclear cells of the type seen in the blood.

Sections were stained with Giemsa as well as hematoxylin-eosin. The lymph nodes revealed a hyperplasia of the reticulum at the expense of the lymphoid elements, with uniform development toward monocytes and macrophages, many of the latter containing pigment, vacuoles, and phagocytosed red blood cells. Mitoses were numerous. Crowding the sinuses were primitive cells with deep blue cytoplasm and light, large, round or slightly lobulated nuclei with nucleoli.

In the spleen, too, there was reticular hyperplasia and obliteration of the follicles. The prevalent cell was of primitive reticular type with round hypochromatic nuclei and a variable number of nucleoli. Hemosiderin in a fair number of these cells underlined their character of macrophages. Extramedullary hematopoiesis was marked and megakaryocytes numerous. No granulocytes could be positively identified.

In the liver, the portal areas and sinusoids were extensively infiltrated with young reticular cells identical with those in other reticuloendothelial tissue (fig. 35).

Monocytic leukemia was also diagnosed in a 6-year-old tom with a necrotic bite wound, profound anemia, jaundice, fever, and moderate enlargement of the liver, spleen, and some nodes. The total white blood cell count was in the normal range (8,000/cmm.), but the blood film disclosed 29 per cent monocytes and a few monoblasts (fig. 36, 37). Marrow and tissue changes were similar to those in the other cat.

In the interests of differential diagnosis, it should be mentioned that on the basis of blood films and physical findings, such as moderate enlargement of nodes, liver, and spleen, monocytic leukemia was strongly suspected in several other anemic cats. The hyperplasia of reticular tissue found on microscopic study did not appear, however,

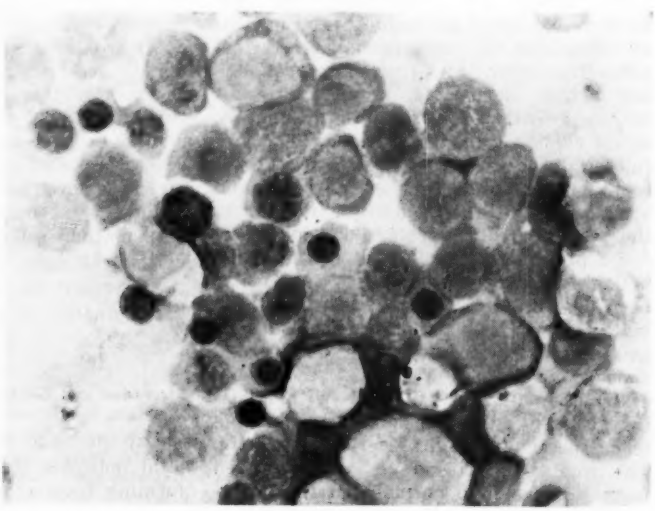

Fig. 34—Monocytic leukemia, marrow, same cat as in figure 33. Large "blasts" with a rim of cytoplasm are mingled with a large number of smaller nucleated red blood cells and a few very lightly staining young myeloid cells. Wright's stain; x 950.

to be malignant, the term "reticuloendotheliosis" perhaps best describing the changes.

Myeloma

Myeloma is a tumor of bone marrow, and its constituent cells may be plasma cells, or cells of the lymphoid, myeloid, or erythroid series. When it is generalized ("multiple myeloma"), tumorous nodules accompanied by absorption of bone give an impression, radiographically, of "punched out" bone destruction. Sometimes, however, the tumorous growth is diffuse, so that its nature is not evident radiographically or anatomically ("diffuse myelomatosis"). Occasionally the myeloma cells enter the blood and are recognized in stained films. Globulin in the plasma is often increased, and Bence-Jones protein may be present in the urine.

One instance of this rare disorder has been described in the cat.[61] Diffuse myelomatosis with hematologic manifestations and involvement of the liver, spleen, and nodes was encountered in a 7-year-old castrated male silver Persian with a history of dental disease, stomatitis, inappetence, persistent high fever, and anemia. The total white blood cell count ranged from subnormal to normal, but the differential count disclosed a substantial proportion (10%) of large mononuclear cells in which the nucleus was more reticular and the cytoplasm less blue and uniform than usual in a young lymphocyte. Some resembled reticular cells or monoblasts; others, myelocytes. Many had eccentric nuclei. In supravital preparations, the cells most nearly suggested pathological, relatively agranular myelocytes. Because of marked rouleaux-formation and precipitation of fibrin a myeloma was suspected. Although no Bence-Jones protein was present in the urine, electrophoretic studies indicated a substantial increase in *gamma* globulin. Examination of the marrow disclosed tumorous foci in many of which there were central areas of undifferentiated cells, some of reticulum type, with maturation on the periphery in the direction of lymphocytes and plasma cells. In the lymph nodes, plasma-cell infiltration was prominent; in the liver and spleen, the malignant cell was predominantly of reticulum-cell type.

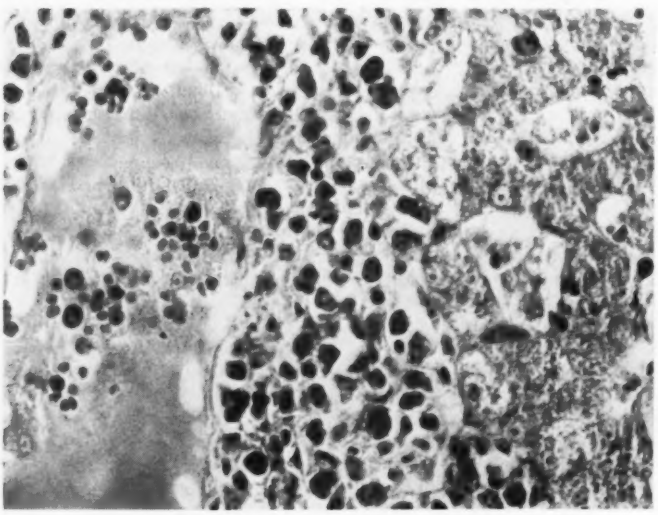

Fig. 35—Monocytic leukemia, liver, same cat as in figure 33. Extensive periportal infiltration of the liver with cells of reticular type. Large mononuclear white blood cells and nucleated red blood cells are present in the lumen of the vein. Giemsa's stain; x 400.

Discussion and Conclusions

Experience at the Angell Memorial Hospital indicates that malignancies of blood-forming tissues are a prominent cause of

fatal illness in the cat and occur far oftener in this species than in the dog. From this hospital's group of over 150 cases, as well as from published reports and studies, it is clear that in the great majority of cases the disturbance is of lymphoid type, ranging from single tumors, through multiple or generalized neoplasms, to leukemia with severe bone marrow involvement and hematologic manifestations. Of striking frequency and clinical importance in the cat are lymphosarcomas and reticulum-cell sarcomas of the anterior mediastinum, intestine, and kidneys. Also characteristic of this species are highly malignant diffuse tumors, of reticulum-cell variety, of the spleen and liver, leading to tremendous enlargement of these organs, sometimes with manifestations of pancytopenic hypersplenism or liver failure. While frank leukemias in which the lymphoid cells occur in large numbers or primitive forms in the circulating blood constitute only a small proportion of the group, it is often possible to gain a clue to the underlying disorder by finding in the blood, even in the presence of a leukopenia, a few cells of primitive lymphoid or reticular type. Severe anemia and thrombocytopenia are often present too.

Myeloid leukemias occur occasionally, the most constant signs being anemia and enlargement of the liver and spleen, with increased numbers or abnormal proportions of young or primitive cells of the neutrophilic series in the circulating blood.

Among the rarer leukemic disorders, several instances of basophilic or mast-cell leukemia are on record, and eosinophilic and monocytic leukemia have also been ob-

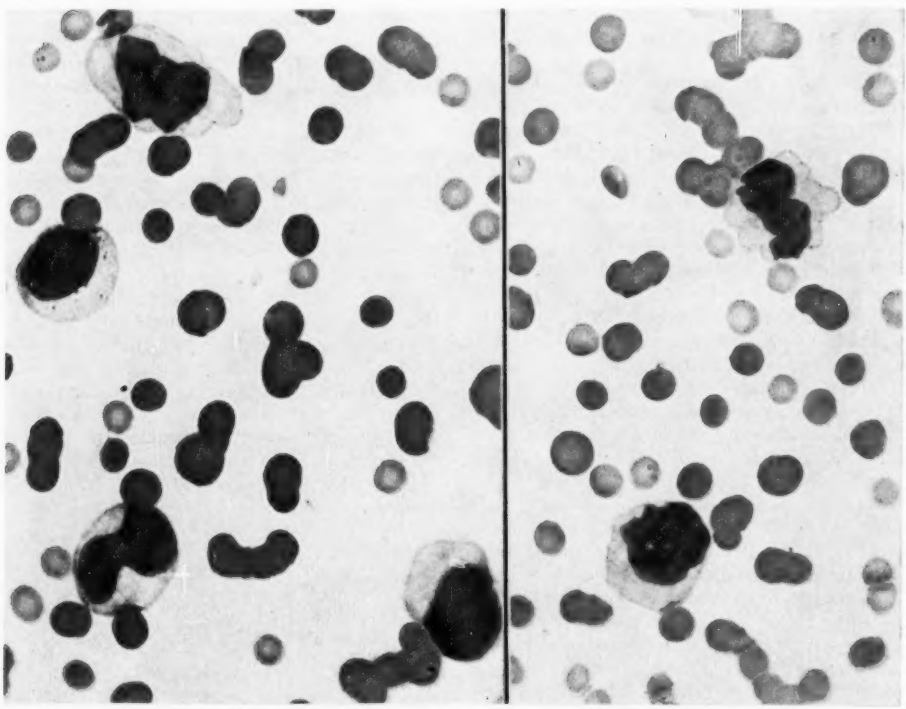

Fig. 36—Monocytic leukemia in a 6-year-old male; w.b.c., 8,000, Hb., 4.5 Gm.; 29 per cent monocytes. Blood—Of the five monocytes in both pictures three are notable for their coarse, irregularly shaped nuclei and ruffled cytoplasmic outlines. One large lymphocyte is present (left), with dense oval nucleus, clear pale cytoplasm, and azurophilic granules. Wright's stain; x 950.

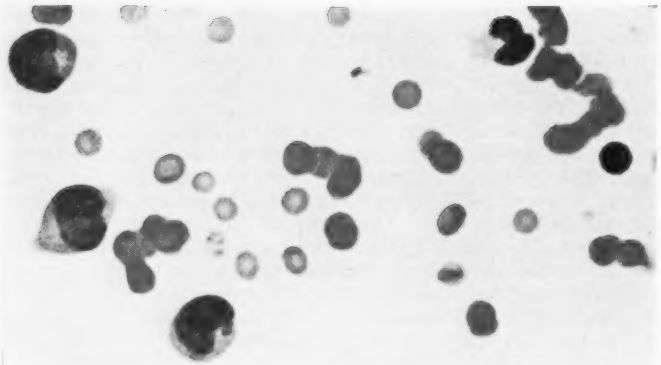

Fig. 37—Monocytic leukemia, same cat as in figure 36, lower power. Three monocytes contrasted in size with a small lymphocyte and a segmented neutrophil.

served, as well as a case of reticulum- and plasma-cell myelomatosis.

There can be no doubt that blood malignancies of all these types go unrecognized far oftener than not, in the absence of the clinical and necropsy studies required for diagnosis.

Certain signs should alert the clinician to the possibility of a malignancy of blood-forming tissue: enlargement of nodes, spleen, or liver; or tumor formation in certain sites of predilection, such as the anterior mediastinum, kidneys, and intestine. (Here it should be cautioned that a large spleen, which may also occur in "infectious" and hemolytic anemia, is an indication for thorough laboratory studies, not precipitate splenectomy!) Anemia, more frequent possibly in the cat than in any other species of domestic animal, is in a significant number of instances associated with leukemias and lymphoid tumors, and is therefore always an indication for blood studies. Even when pathognomonic features of leukemia are not present in the blood, a persistently low hemoglobin, perhaps with leukopenia or thrombocytopenia, may be reason to suspect a "sub-" or "aleukemic" malignancy, and a thorough search of blood films is often rewarded by the finding of a tell-tale "blast" or reticulum cell. Fever is present in many leukemias and is often associated with other signs which may for long throw the clinician off the track—a necrotic, nonhealing wound or a refractory mouth infection. In short, the obvious and clear-cut leukemias are in the minority, many masquerading in the guise of other conditions or betraying themselves by signs so nonspecific that they are recognized only after painstaking diagnostic studies, after time clarifies the picture, or at necropsy.

Why the cat, more perhaps than any other domestic animal, should be prone both to anemia and to leukemic disorders is at present not clear. Although the relationship may be purely coincidental, several cases in which episodes of anemia antedated such malignancies suggests the tempting hypothesis that the one problem may be related to the other, profound anemia perhaps in some way stimulating the blood-forming tissues to abnormal activity. In the past histories of many, many more cats, perhaps again by coincidence, infection of one type or another appeared prominently. While further clinical studies will undoubtedly attest to the frequency and variety of leukemias and related disorders in the cat, it is likely that their etiology and possible relationships to factors such as infection and anemia will be clarified only by experimental investigations.

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Corneal Ulcers in Cats Eliminated by Denervation

Corneal ulcers developed in all of 10 experimental cats, in which the trigeminal nerve was completely interrupted. When the stellate ganglion and the nictitating membrane were removed prior to cutting the trigeminal nerve, a beneficial effect was observed in all animals, and only 1 showed evidence of a lesion.

The specific influence of the sympathetic nerves in this protective mechanism is not known, but it is believed to be linked with increased vascularity and fluid permeability and better regenerative powers in the damaged cornea.—*Proc. Mayo Clinic, 34, (1959): 474-478.*

Griseofulvin for the Systemic Treatment of Dermatomycoses

Two hundred patients with superficial fungous infections were treated with griseofulvin. Dermatomycoses of the skin, nails, and hair were almost invariably cleared with the use of this drug.

Griseofulvin appears to have low toxicity for man. No specific contraindications to it are known. Periodic blood counts should be taken during prolonged therapy. A dose of 1.0 Gm. per day is recommended until clinical clearing has occurred—about 3 weeks for skin lesions, 4 to 6 weeks for foot and hand lesions, 3 weeks for Microsporum infections of the scalp, 6 weeks for nonfluorescent infections of the scalp, 4 months for lesions of the fingernails, and 6 months for lesions of toenails. Mycologic and bacteriologic studies are desirable for accurate diagnosis and follow-up.—*Harvey Blank et al. in J. Am. M. A., 171, (1959): 2168-2173.*

In Small Dogs

Vaccination Safety

Using Bivalent Modified Live Virus Vaccine

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George J. CHARLEBOIS, D.V.M.

BECAUSE the safety of a recommended 2-cc. dose of reconstituted modified live canine distemper and infectious hepatitis virus vaccine for small breeds of dogs has been questioned in our practice, only half the 2-cc. dose of vaccine has been used. No specific vaccination recommendation regarding administration and effectiveness of partial doses of this vaccine for small breeds has been obtainable.

When modified live infectious canine hepatitis virus vaccine of porcine origin combined with canine distemper modified live virus vaccine* became available, 71 small-breed pups were vaccinated with a 2-cc. dose on an experimental basis in order to ascertain the safety of the product. Those vaccinated included 6 Pomeranians, 12 Chihuahuas, 6 "toy" Terriers, 8 "miniature" Dachshunds, 5 Pekingese, 1 "toy" Cocker Spaniel, 2 "toy" Pugs, 10 Toy Poodles, 8 Miniature Poodles, 2 "toy" Boston Terriers, and 11 mixed breed toys. Ages varied from 7 to 14 weeks; weights varied from 1½ to 4 lb.

Inoculations were made subcutaneously in the flank since owners of small dogs indicated that they do not like their pets vaccinated in the neck area. Homologous anticanine distemper and infectious canine hepatitis serum, 1 cc./lb. of body weight, was given on the side opposite that where the vaccine was given. Some dogs were observed for several hours at the hospital following vaccination. All owners were instructed to call immediately if any untoward reaction occurred.

No reactions of any kind occurred except for slight tenderness at the site of serum injection and at the inoculation site of the vaccine. Aspirin tablets were dispensed and each owner instructed to administer a 0.5-gr. tablet at four-hour intervals if the pup cried when the flank was gently massaged. This program satisfied the owners and kept the pups contented. The recommended 2-cc. dose of this vaccine appears to be completely safe for vaccinating pups of small breeds.

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*CYVAC is produced by American Cyanamid Co., Pearl River, N.Y.

Life Span of Red Blood Cells in the Dog

The average life span of red blood cells in pups and adult dogs is 98.8 and 105.2 days, respectively. The life span of red blood cells is approximately the same whether they are transfused by intravenous, intraperitoneal, or intramedullary routes. The life span of red blood cells in dogs with a subclinical and clinical hookworm infection was 92.9 and 21.7 days, respectively. This was undoubtedly due to losses of blood through the parasites.—[*Carl H. Clark and C. H. Woodley: The Effects of Certain Diseases and Transfusion Methods on the Life Span of Red Blood Cells. Am. J. Vet. Res., 20, (Nov., 1959): 1069-1071.*]

Teat and Udder Pathology — Treatment and Surgery

Open Teat Sinus Surgery

for Correcting Teat Occlusions — Part II

James H. STEERE, D.V.M.
Kenneth M. MOODY, D.V.M.
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IV. Treatment of Miscellaneous Teat and Udder Abnormalities

Contusions.—Contusions of the teat with badly macerated and swollen tissue are the most difficult of all teat injuries to treat satisfactorily. Cannulas or dilators are necessary for milking the quarter, and prophylactic treatment for mastitis is imperative. Swelling may be reduced and milking made easier by soaking the teat twice daily in a warm Epsom-salts solution. An antiphlogistine poultice, applied after each milking and covered with a loose bandage, will further aid in reducing swelling. In severe cases, however, it is best to infuse the quarter with mastitis ointment, allow the quarter to go dry, and reëstablish patency, if necessary, on the subsequent lactation.

Leaking Teats.—Fistulas resulting from lacerations are best closed during the dry period. Very small openings can sometimes be closed by cauterization with caustics, such as butter of antimony or silver nitrate. In large fistulas, however (and we believe even with very small ones), it is better to use the surgical approach, excising all old scar tissue with an elliptical incision through to the sinus, and closing the fresh tissue as described in Part I. The following is a useful variation[5] on this method. First remove the scar tissue around the fistula, make an "H" incision in the skin with the bar of the H over the fistula, bring the two skin flaps between the parallel incisions together and suture them over the fistula, then suture the uprights of the H.

Leaking sphincters from injuries, old age, etc., may be helped by injections of 2 to 3 minims of Lugol's iodine at four points around the teat papilla, using a tuberculin syringe and needle. The iodine will cause counterirritation with resultant cicatrix formation and help "tighten" the sphincter. Repeated injections can be made to effect and, though it seems impossible to stop some teats from leaking, some benefit is usually derived from the attempt.

Accessory Teats.—These are best excised when the heifer is a calf, when they are simply cut off with a pair of scissors or knife and no suturing is required. This is best done when the calves are dehorned. If six teats are present, usually the posterior set are accessory—on rare occasions, the middle pair. If it is impossible to determine which are the accessory teats, allow the udder to mature before attempting their removal. The dry period is the best time for removing accessory teats from the mature gland, especially where the accessory teat has a functional gland. Desensitize the base of the teat with procaine solution (1 or 2%), excise it with an elliptical incision (usually a pair of heavy curved scissors are best), and take 2 to 3 simple interrupted sutures with catgut. If the accessory teat has a functional gland it is sometimes best to have the owner perma-

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nently dry the gland two to three weeks before surgery.

If the accessory teat is attached to a functional one, the excision should be done carefully in order to avoid opening the functional teat sinus. On rare occasions, the accessory teat looks more like a secondary orifice of the primary gland, and a differential diagnosis can be made by infusion of a dye into its opening to see whether the dye mixes with the secretion of the functional gland.

Except for aesthetic reasons, it would be unimportant to remove these accessory teats, but if the accessory teat has a functional gland, mastitis often develops and, if acute, may cause a production loss due to illness. In severe cases, where the secondary and primary glands are in juxtaposition, the infection sometimes erodes through to invade the parenchyma of the functional gland.

Drying Glands.—Permanent destruction of the alveolar tissue can usually be accomplished with 1 to 3 infusions of 50 to 500 cc. of 3 per cent silver nitrate, the wide variation in dosage depending on the size of the gland.

Advice to the dairyman on drying the gland for the normal 60-day rest period must be guarded since no single formula is satisfactory. First of all, this is a good time to start a simple mastitis control program by advocating the CM test on quarter samples before the gland is dried, keeping a record of the positive quarters, with subsequent treatment when the gland is completely dry and atrophied.

Generally, if there is little mastitis present (CMT 1 or less) and the cow's milk production has been on the decline, it is safe to simply stop milking the gland. If mastitis is present (CMT 2 or 3), this method is likely to cause a flare-up, and immediate treatment of these quarters and a more gradual drying of the gland is required. In some animals, this is difficult because of their continued high milk production. Difficulty in stopping lactation is often an indication of nonpregnancy, and some such cows, because of natural longevity of high production, are worth treatment and rebreeding—if they have not already been "dried off" by an unknowing owner. (You can render such dairymen a real service by teaching them fetal ballotment so they are able to "feel" the calf.)

Teat and Udder Amputation.—In gangrenous mastitis it is important to establish adequate drainage to help relieve the concomitant toxemia. If the gangrene does not involve the teat, excise the affected tissue, which is easily demarked from healthy tissue by being insensitive and ischemic. While it is empirical, we believe that the further spread of gangrene is retarded by intravenous injections of oxytetracycline, ringing the gangrenous area with deep parenchymal injections of streptomycin, putting a fomentation bag on the udder and applying alternate hot and cold packs. If the teat does not become involved, it is sometimes possible to restore the quarter to functional activity in the present lactation or, with subsequent dry treatment in the next lactation. If the teat becomes involved, of course, it should be amputated to facilitate drainage.

If the cow survives the acute stages of the mastitis, but loses one or more quarters, it is then practical and economical to amputate the udder and salvage her for beef. A cow recovering from gangrenous mastitis has almost constant low-grade fever from the absorption of toxic material from the sloughing tissue; thus, her appetite and weight gain are retarded, and it takes two to four months for the affected tissue to slough and enough healing to occur to be sure the cow will pass slaughter inspection. Following udder amputation, a cow rapidly regains a normal appetite and weight, and it takes only two to four weeks for adequate healing to make her acceptable for slaughter.

A complete description of the surgery can be found in any veterinary surgery manual. Briefly, the cow is anesthetized and placed in ventral recumbency; the skin of the udder is incised about midway between the base of the udder and the teats (depending on the area of gangrene); the blood vessels, mainly the external pudendal and abdominal veins, are ligated; the gland is dissected away from the prepubic fascia; and the skin is sutured so as to avoid cavities.

Teat Papillomas.—These are, from the point of treatment, of two types: surface and pedunculated. The surface wart is best treated with Fowler's solution of arsenic, 1 tablespoon twice daily in the grain for 2 weeks, withheld 1 week, and repeated as required. Response is usually seen in about 3 weeks. If desired, local application of any number of proprietory compounds may

be used in addition to the Fowler's solution.

The pedunculated warts are best removed from the teat by surgery or ligation when the cow is dry. Lately we have been using electrocautery after first tranquilizing the cow. So far the results, even on teats being milked, seems favorable. Pedunculated warts often occur in the teat orifice and are precursors of mastitis. They are best removed by deep surgical excision, followed by the aftercare recommended for an injury of the teat papilla.

Pseudocowpox.—This disease, apparently of virus origin, causes much misery in many herds between the fall and spring equinoxes. Unlike true cowpox, there are frequent recurrent attacks. The teats are sensitive, the cow objects to being milked, and her production falls. If the disease, which begins with blisters, involves the end of the teat, mastitis is the frequent sequela. Treatment consists of applying a desiccating agent, such as equal parts of 7 per cent iodine and glycerin, during the blister stage, and a good bactericidal emolient during the ulcer and healing phase. Prophylactic mastitis treatment should be used whenever the lesions involve the teat papillae. Though empirical, sulfur sublimate[6] (flowers of sulfur), 1 teaspoon per head daily in the grain or on the silage during the fall and winter months, seems to have a decided inhibiting effect on the incidence and severity of the disease.

Psychic Agalactia.—The problem of general agalactia, involving mastitis, malnutrition, systemic illness, heredity, udder hypoplasia from endocrine insufficiency, etc., is too complex to discuss here. However, psychic agalactia which occurs frequently, usually in fresh heifers, is easily treated. The causes center primarily around maternal distress from having the calf taken away, the strange environment of the milking barn, and udder manipulation by man and machine rather than by offspring. Oxytocin, 25 mg. intravenously, or 50 mg. intramuscularly, will usually bring about milk let-down, and if repeated each milking until the psychic distress has worn off, the condition will disappear. The ataraxics, since they act directly on the central nervous system, relieve the psychic distress promptly, and though some animals are refractory to tranquilizers, usually 50 to 100 mg. will maintain a euphoria over two milkings, enough time to clear the condition in the majority of newly fresh heifers.

Parturient Udder Edema.—This condition, which occurs most frequently in heifers, should be relieved if possible because of the danger of mastitis and udder breakdown. Mastitis frequently develops in these udders because of the poor circulation and because milkers have a tendency to leave the machines on these udders too long, the edema masking the empty quarters. The following measures are recommended: Withhold concentrates and rich hay (though this will increase the possibility of ketosis occurrence); use a fomentation bag with cold water or ice packs; vigorously massage the udder with a mild counterirritant, such as white liniment; milk more frequently and, in severe cases, resort to preparturient milking; use oxytocin to elicit more complete milk letdown; increase exercise; and give diuretics, though these are of doubtful benefit owing to the cost and frequency of required dosage.

Udder Hemorrhage.—This occurs most frequently following an injury or in the congested udder of the fresh heifer. The condition, caused by capillary rupture, usually clears in seven to ten days. In severe or unresponsive cases, however, the following treatment will help: Give the cow a dry, non-milk-producing feed; hand milk the affected quarter(s); give injectable vitamins C and K; and use parenteral coagulants. Most of the proprietory drugs are too expensive for large animal use. However, the following intravenous one-dose treatment is within reason and usually begins clearing the milk within 24 hours: 2 drams of 38 per cent formalin in 250 cc. of sterile distilled water or sodium chloride solution.

Udder Ulcers.—So-called "summer sores" are ulcerated areas in the cleft between the front two quarters where the udder joins the body. The ulcers begin with irritation from rubbing of the two quarters due to the pressure of the full gland. Heat and flies add to the irritation until a foul-smelling ulcer forms from epithelial erosion and secondary bacterial infection. The ulcer may be held in check by first washing it thoroughly with strong, hot chlorine solution, followed by the twice daily application of a desiccating agent such as iodine and glycerin. Twice weekly applica-

tion of screwworm repellent during the warm months will discourage fly irritation. Cold weather will bring relief. Permanent cure can be obtained only by surgery. An elliptical section of skin including the ulcer is excised and the void is closed by sutures. The necessary tension used in closing the gaping wound eliminates the former cleft between the quarters so that recurrence of the ulcer is rare.

Udder Breakdown.—This condition is caused by the loosening, stretching, and rupture of the median and lateral suspensory ligaments of the udder in the progressively maturing cow. In severe cases, the gland has an almost "Gibson girl" appearance due to the narrow-waisted separation of the pendulous udder from the body. While treatment of cows with extreme cases may be hopeless, the use of an udder support may be warranted occasionally. In high-producing animals, and especially in those in which congenital predisposition may be suspected, ovariectomy at 60 to 90 days postpartum may be advised.

While unrelated to teat and udder surgery, ovariectomy is important, since cows with progressive udder breakdown can be kept for one to five years of economic production without parturition and its resultant dangers to the already sagging gland. Even if the cow's lactating life were not prolonged through ovariectomy, the resulting absence of estrus and its associated hyperactivity would reduce injuries to such an udder.

Discussion

An attempt has been made to promote a newer concept and approach to bovine teat surgery and to the problems of miscellaneous udder and teat anomolies. The routine use of the visual open-teat technique in removing mechanical obstructions of the teat canal system is recommended rather than the old approach through the streak canal, an approach which is blind, crude at best, and many times completely ineffective.

In almost all cases, this open-teat approach for the removal of teat spiders and other mechanical obstructions within the sinus has been completely successful in establishing patency of the canal system, and no recurrences have been observed on subsequent lactations provided the quarter is cleared up by treatment during the interlactation period. We feel that in almost all teat obstructions above the streak canal, unless minor, open-teat surgery is preferred because of the visual surgical field it offers, the over-all time-saving, and the resulting functioning of the canal and lactating gland.

Most surgery through the streak canal should be limited to pathologic conditions of the canal, except for minor polyps, plugs, and flaps farther up in the sinus which can usually be extracted through the streak canal with alligator forceps. The use of various types of teat reamers, most of which resemble something out of a medieval torture kit, is questionable because of the trauma they inflict to the delicate mucosa. The number of types of such instruments belies their effectiveness. If a bistoury or alligator forceps will not relieve sinus impatency, it is best to resort to the open-teat technique.

Though we have had little experience with the German method of teat suture and none with the skin implantation outlined previously, they both warrant investigation. The suturing technique advocated[3] would greatly simplify the one used by the authors, and the method[4] of filling in areas of denuded teats with skin implants could take over where the method described here leaves off. The limiting factor in both these procedures is the bandaging advocated; unless it can be simplified, this restricts these methods to the institutional clinic since drawn-out aftercare precludes their routine use in commercial dairy herds.

Investigations comparing the German suture technique with the one advocated in these articles, have revealed that there is some difficulty in judging necessary suture tension which will both bring about adequate closure and allow for postsurgical swelling. Also, suture removal seems unduly difficult. One reason for the difference in results may be that while this suture method is primarily advocated on the dry quarters, we are trying it on quarters which were milking and subjected to daily manipulation.

The conditions described in section IV are commonly dealt with by the experienced dairy practitioner. However, because

the veterinary schools have more of the bizarre than the mundane in their clinics, most new graduates are poorly equipped to handle these most common anamalies or to offer good advice on their treatment. Since there is little in the literature to help them, we have tried to fill some of this void. The treatments listed in the last section are largely empirical and await more thorough investigation; if they have raised more questions than they have answered, insertion has been worthwhile.

Summary

The surgical opening of the teat sinus for the removal of various mechanical and congenital obstructions, such as teat spiders, is described in detail. The teat is incised to the sinus, the mucosa is everted through the incision, and the obstructions are visually excised. The mucosa is closed with a continuous suture; the submucosa with a continuous mattress suture; and the skin with an interrupted vertical mattress suture.

Other teat and udder anomalies which require surgery, or are subject to treatment, are covered in some detail, as is the importance of pre- and postsurgical care of the teat, especially with regard to mastitis prevention following surgery or trauma.

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Foot-and-Mouth Disease Carrier Cattle

Research workers in Holland observed that a rather large proportion of cattle which had recovered from foot-and-mouth disease still harbored the causative agent in the saliva during a period of several months. The virus was demonstrated in material collected from the esophagus by inoculation into unweaned mice or susceptible cattle. After contact with clinically affected cattle, vaccinated animals developed a similar carrier state without having shown signs of the disease.

Susceptible oxen kept in contact with such carriers remained unaffected, even if the oral cavity was swabbed with infective saliva. No cases of foot-and-mouth disease occurred in vaccinated or unvaccinated cattle or in unvaccinated pigs, if such animals were introduced into a herd known to contain carriers or if they were kept on the same premises with such animals. Thus, in cattle carrying foot-and-mouth disease virus, a continual formation of small quantities of infective material must be taking place. Purely mechanical carrying seems highly improbable.—*J. G. van Bekkum et al. in Tijdschr. voor Diergeneesk. (Oct. 15, 1959): 1159.*

Effect of Antibiotics on Immunity

Of the various ways in which antibiotics can affect immunity unfavorably, it was shown that with chloramphenicol these three are valid: (1) there is an effect on the host; (2) there is an effect on the antigenic constitution of the infecting agent; (3) there is a reduction of antigenic stimulation as a result of considerable diminution of the numbers of infectious bacteria.

The inhibitory action exerted by antibiotics on the formation of antibodies must be taken into account when interpreting laboratory findings. Although serologic tests often provide valuable information for the clinician, the establishment of weak immune reactions or even complete absence of antibodies can be confusing, especially if specific infection has been suspected on the basis of clinical or bacteriologic examination.

The relationship of antibiotics to immunity indicates that antibiotherapy should be considered on a new basis: the antibiotic subjugates the infection, but body defenses assure the recovery. In fact, known experimental data show the importance and efficiency of specific vaccination whether for prophylaxis or associated with antibiotic therapy.—*Ann. Inst. Pasteur, Nov., 1959.*

The Effects of Antibiotics on CRD in Chickens

Weight gains produced chickens in experimentally inoculated with chronic respiratory disease virus and then treated with either chlortetracycline or oxytetracycline were higher in all but one group than weight gains obtained in nontreated-inoculated birds. Groups treated with oxytetracycline plus terephthalic acid showed slightly higher weight gains in some cases than those treated with oxytetracycline only. Noninoculated groups achieved highest weight gains. The antibiotics exerted only slight inhibition on signs and lesions of CRD.—*Avian Dis., 3, (1959): 457.*

Antibiotics for Malignant Edema

Recovery of a horse from malignant edema due to *Clostridium novyi* was obtained from large doses of penicillin and oxytetracycline, in conjunction with local treatment (incision of the edematous swellings and irrigation with hydrogen peroxide). This is thought to be the first case of successful antibiotic treatment of malignant edema in the horse.—*Wien tierärztl. Monatschr., 45, (1958): 650.*

Penicillin-Streptomycin Combination Questioned

Although penicillin and streptomycin may be useful when given simultaneously in special circumstances, it is difficult to find any specific indication in which any fixed dosage ratio of these two drugs could be justified for inclusion in a single package. The lure of convenient packaging apparently has encouraged use of this fixed combination as a preferred prophylactic and therapeutic agent rather than a specific need for this product combination.

Treatment with this combination is often maintained for long periods in the mistaken notion that it may be preventing infections and that it is innocuous. Under such conditions, it is not surprising that serious and irreversible hearing loss due to the streptomycin fraction is encountered in these cases. This ototoxic complication often becomes manifest or fully developed only weeks or months after the drug treatment has stopped and relation to its use is thus obscured.—*Editorial in New England J. Med., 261, (Sept. 24, 1959): 666.*

A Practice Builder

Routine Pregnancy Examination

in Beef Cattle Practice

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EXAMINATION of the reproductive organs, per rectum, to determine pregnancy in cows is a veterinary service that is finding increasing demand by beef cattlemen. The information gained by this procedure yields immediate monetary rewards to the cowman, because he can decide whether to cull his "open" cows in the fall and either rebreed them, hold them over, feed them to a desirable grade, or sell them on the market, depending on value and availability of feed, market price, and herd management.

Dairy farmers lose $1.00 a day for every cow that has not settled. A program of regular weekly or monthly examination by a veterinarian for pregnancy or breeding problems in the herd could alleviate this expense.—*Kansas Stockman, 45, (1959): 44.*

With this information, he can anticipate his calf crop and can segregate his cows in groups according to probable calving date. In general, he can operate more efficiently and does not have to wait until spring until he can observe the signs of impending parturition. Should he buy replacements, subject to pregnancy examination, he can avoid buying open cows he may not wish to acquire.

A trader can sell a "preg" tested cow at a higher price than one whose status is indefinite. Consequently, he will employ this veterinary service whenever he sees a chance of increasing a cow's value. Such a client is interested only in learning whether a cow is pregnant and, if so, when calving is expected. Furthermore, he is intolerant of any error of diagnosis and avoids the practitioner whose diagnoses are not reputedly accurate. Hence, it behooves the veterinarian to do careful work and be certain of his diagnosis.

In the case of the cowman, a critical inspection of every cow is usually appreciated. During the course of examination, there is ample time for discussion and answering clients' questions. Disease control programs can be aired. Local infections and abscesses may be treated or marked for subsequent treatment. Ectoparasites may be diagnosed. In our area, the opportunity to see the eyes of a Hereford herd almost always reveals the need for surgery, because neoplasms of the eye are a serious problem. Their early detection, often made possible by examination of the cow at the time of pregnancy diagnosis, gives the owner a chance to make himself more money by investing in further veterinary service.

Pregnancy testing can continue to be a valuable service rendered by veterinarians if handled wisely, but it could be appropriated by lay technicians. To a large degree, who does the pregnancy testing will be decided by fee policies. In many parts of the country, a growing number of artificial insemination technicians are anxious to engage in pregnancy testing. Ranchers have stated that there are itinerant laymen who will do pregnancy testing for 25 cents and less per head. So, fees

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should be attractive to the cattleman as well as the veterinarian. Experience indicates that a flexible fee policy based on circumstances is satisfactory. If the number of cows to be examined is comparatively small, a fee of $1.00 per head up to 25 is usual. If more are involved, a reduction may be in order. If a ranch herd is to be examined, a charge figured on a time basis has advantages; $15 an hour or $125 to $150 per day may induce the rancher to fix up his corral and chute to facilitate the work, increasing the number that can be examined in a given time and making the work easier for the veterinarian.

A sleeve-length obstetrical glove and a can of lubricant powder is the only special equipment needed. If a large number are to be examined, an old well-stretched rubber glove will prove to be much more comfortable than a tight new one. Plastic gloves may be used, but their durability is variable. The sleeve-length glove is worn on the examining hand while a regular wrist-length rubber glove is worn on the other hand.

A routine procedure should be established. For example, I grasp the cow's tail with one hand as the examining hand and arm, well-moistened and lubricated, is inserted into the rectum in stiff-arm fashion, coincidental with the forward motion of the body. If feces are dry and hard, they should be removed; if soft, removal is usually not necessary. As the fingers reach the brim of the pubis, a sweeping motion locates the cervix or uterus or detects its absence. If present, it can quickly be followed to determine the stage of pregnancy. Determination of the trimester of pregnancy usually provides enough information to satisfy the owner's needs.

If no structure disturbs the continuity of the brim of the pubis, the curled uterus is encountered by the palm of the hand as it is withdrawn. Occasionally the uterus is in the dorsolateral area of the pelvis, with a left horn fetus sometimes hidden under the pelvic extremity of a rumen distended more than we prefer.

To ease examination and handling, withholding feed for 24 hours is advantageous.

Immunization of Horses Against Influenza Using Live Virus

A living Hh-2 strain of equine influenza virus was used to actively immunize 5 susceptible 4-year-old Thoroughbred mares. Immunization was achieved by intrapharyngeal inoculation with 2 Gm. of emulsified liver tissue from aborted fetuses. A mild temperature rise lasting one to three days followed. Production of antibodies reached their peak in the blood between the second and fourth weeks and disappeared by the twelfth month. It was approximately eight weeks after immunization before virus excretion ceased.

Fourteen months later, the same inoculation procedure was repeated on 3 of the immunized mares and 1 control, one to two months before they were due to foal. The control aborted while the immunized mares did not.—*Vet. Bull., 29, (1959): Item 2096.*

Bloat Caused by Rumen Bacteria

A relationship between the number and types of bacteria in the rumen and the degree of bloat in cattle in feedlots has been established in studies conducted by USDA scientists.

Bacteria in the rumen of cattle before bloat consist in part of the normal small single or paired streptococci and rod-shaped bacteria. But in the early stages of bloat, brought about by feeding rations high in starch supplemented by ample protein, there are large numbers of lactic acid-producing starch-fermenting streptococci surrounded by capsule-like gelatinous material, and a second type of large streptococci which utilize the lactic acid. These bacteria, *Streptococcus bovis* and *Peptostreptococcus elsendii* increased in numbers as bloat increased in severity.

A growth characteristic of these bacteria is their production of a viscous, ropy slime long considered a factor in feedlot bloat. The abundance of slime-producing organisms found during bloat tends to verify this theory. The slime may alter viscosity of the rumen fluid and contribute to trapping of fermentation gases. Resulting frothy material blocks the release of gases causing typical swelling of bloat.

Laboratory cultures of *P. elsendii* did not produce large amounts of slime, but did produce large quantities of gas from lactic acid and carbohydrates. This finding suggests that these bacteria augment bloat by breaking down lactic acid. Another factor contributing to bloat may be the extremely heavy cellular growth of Streptococcus organisms which form a filament-like mat during bloat.—*USDA Press Release, Nov. 5, 1959.*

Estrogen Toxicosis in Pigs

A group of 30 pigs, 8 to 11 weeks old, showed varying unthriftiness although their rations seemed adequate and free of mold. Although much too young for estrus, all the gilts had congested and edematous vulvas and, for at least 14 days, had acted as if they were in heat. Both males and females showed enlarged nipples and a relaxed perineum and anal sphincter. Their appetites were variable, as if the gilts were preoccupied with estrus. No significant abnormalities were found on necropsy of 2 such pigs.

It was learned that heads of capons, which had been boiled into a soup, had been fed to these pigs regularly for six weeks; other groups not fed this soup were normal. Apparently, the estrogen residue from 15-mg. pellet implants per capon were responsible.

When the capon heads were withheld, manifestations of estrus abated in a week, the vulval swellings subsided in a month, and the general conditions of the pigs improved.—*Austral. Vet. J. (March, 1959): 90.*

Diethylstilbestrol in Dairy Cattle

Thirty-one dairy cattle in various stages of gestation were used to determine the effect of orally administered diethylstilbestrol on conception, gestation, and parturition. Diethylstilbestrol was included in a 17 per cent ration at levels of 0, 5, 10, 15, and 20 mg./1,000 lb. of body weight per day. All animals received alfalfa hay and sorghum silage.

Throughout the experiment, no difficulty in conception could be observed. All cows killed prior to parturition yielded apparently normal embryos. Cows carrying to full term gave birth to normal calves. However, 3 cows receiving the 20-mg. level aborted.—*J. Dai. Sci., 42, (1959): 1351.*

Ovine Virus Abortion

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THE ETIOLOGIC AGENTS reported to date from ovine abortions in the United States have been *Listeria monocytogenes*, *Leptospira pomona*, Salmonella spp., *Vibrio fetus*, and a virus of the psittacosis-lymphogranuloma group.[2,3] Abortions may result from other infectious, as well as from noninfectious, causes.

One of the most active research projects at the Montana Veterinary Research Laboratory is the study of ovine vibriosis. This study showed that vibriosis was not the only infectious type of abortion present in Montana sheep. During the lambing seasons of 1956, 1957, and 1958, 61 separate epizootics of ovine abortion with an infectious disease history were investigated in Montana. *Vibrio fetus* was the causative organism in more than 62 per cent of these epizootics. However, aborted tissues from 37 per cent of the affected flocks were bacteriologically negative. These negative bacteriologic findings suggested that the etiologic agent had been overlooked. However, it was not until the 1958 lambing season that we were able to identify a virus as at least one cause of the losses among the previously unidentified etiologic agents. Four epizootics that were bacteriologically negative, 3 in Montana and 1 in Idaho in 1958, were proved to be caused by a virus.

Etiologic Findings

Ovine virus abortion was shown to be caused by a virus of the psittacosis-lymphogranuloma group, and it is the same virus as that described in Scotland.[1]

From the Montana Veterinary Research Laboratory (Montana Experiment Station and Livestock Sanitary Board cooperating), Montana State College, Agricultural Experiment Station; paper No. 474, journal series.

Distribution

The reported incidence has been limited to sheep in Scotland, England, Sardinia, Germany, France, Hungary, and the United States.

The virus seems to be widely scattered throughout the United States. Furthermore, the infection appears to have been established for an undetermined time. Although recent work indicates that there is a distribution pattern, sufficient data are not yet available to map the virus' distribution throughout the country.

In 1956, the Rocky Mountain Laboratory of the National Institutes of Health reported applying complement-fixation tests with psittacosis antigen to 734 serums from Utah and Idaho sheep. Positive reactions were present in 7 to 8 per cent. We do not know whether the antibodies represent those of ovine virus abortion since there is a group antigen common to the psittacosis and ovine abortion viruses. However, no other sheep disease that stimulates such antibodies is known to be present in those areas. It is probable that the positive reactions obtained are due to the presence of ovine abortion virus.

During the 1959 lambing season, aborted tissues or serums from ewes that had aborted either in 1958 or 1959 were studied for presence of the virus. Specimens were received from 47 flocks representing 14 states. Study results showed the abortion virus to be present among the sheep of 8 states, with 2 other states having suspicious tests.

Clinical Signs and Pathologic Findings

Abortions occur from mid-gestation through late pregnancy, with the majority in the latter part of pregnancy. The incidence usually does not exceed 5 per

cent although it may reach 25 to 30 per cent. Retention of fetal membranes is common. A vaginal discharge usually occurs for several days. The ewe may lose weight during the course of the disease. The cotyledons are dull-clay to dark-red in color, with or without necrosis, and their consistency appears to be more solid than normal.

Experimentally infected ewes have shown a body temperature rise 3 days after inoculation. The temperature returned to normal by day 8. Maximum temperature ranges were 106.4 to 107.0 F.

The aborted fetus may be mummified, degenerated, or normal, or the lambs may be born alive or dead after a normal gestation period. One lamb of twins may be normal and the other dead. Subsequent fertility of the ewe does not seem to be affected.

Transmission

Experimental.—Pregnant ewes in mid-gestation have been experimentally infected with bacteria-free fetal membranes, fetal tissues, and infected chicken embryo yolk sacs. Nonpregnant ewes, although less susceptible than pregnant ewes, may develop the disease during their first gestation period following oral, subcutaneous, intravenous, or intradermal inoculation of the virus.

Natural.—Normal ewes grazing during pregnancy on pasture where abortions occurred the previous season do not abort. By chicken embryo inoculation techniques, the virus may be isolated from the stomach contents, lung, liver, spleen, and kidney tissues of aborted fetuses. Ewes that have aborted, lamb normally the following year with no evidence of infection. Ewe lambs and unbred ewes, exposed to aborted fetal and maternal tissues at lambing time, may become infected and carry the infection with no outward manifestation until the next pregnancy, at which time they may abort. Thus the infection may be transmitted to a susceptible ewe population.

Without a complete understanding of the mode of transmission of this virus, some sheepmen may become confused regarding the source of new infections. They must realize that ewe lambs may contract the infection and show no apparent signs, yet they carry the virus until they have aborted their first lambs. The course of the disease may result in a lapse of 2 years between the date of infection and abortion.

There is no evidence, either from field observations or experimental work, to show that the disease can be transmitted by the ram.

Differential Diagnosis

In virus abortion and vibriosis, the clinical manifestations are practically identical, and the pathologic changes in both fetus and fetal membranes are grossly similar.

In the case of virus abortion, inoculation of chicken embryos, guinea pigs, mice, or ewes is the most accurate diagnostic method. Microscopic examination of stained smears from the cotyledons usually permits identification of the elementary bodies of the virus.

Complement-fixation or agglutination tests give positive confirmation of virus infection.

Bacteriologic cultures should be prepared to determine the presence or absence of *V. fetus* or other bacterial etiologic agents.

Immunity

Infected ewes rarely abort more than once. In Scotland, a single injection of the vaccine currently in use, a formalin-inactivated chicken embryo yolk sac suspension, is considered capable of producing lifetime immunity. First-lambing ewes or previously unexposed ewes in infected flocks should be vaccinated before breeding. (A vaccine probably could be made available commercially in the United States, provided a sufficient demand were created for the product.)

The rate of abortion decreases rapidly following annual systematic vaccination of all unexposed and first-lambing ewes. Those of us in a position to advise vaccination should emphasize to the client the fact that it is to be used against a specific virus infection and that it does not immunize against any and all causes of abortion.

Control

The disease may be completely controlled by vaccination of all unexposed first-lambing ewes. Complete control may not be possible in flocks previously exposed to the infective agent. Some of the ewe lambs, or older ewes that did not abort from the previous year's exposure, may have been infected before vaccination. As a consequence, vaccinated ewes may abort if infected prior to vaccination.

Since the infection is spread predominantly at lambing time, all of the ordinary preventive precautions should be followed.

The successful control of the disease by vaccination in Scotland, England, France, Germany, and Italy has made regulatory control measures unnecessary. In those countries, it is not classed as a reportable disease.

Treatment

No specific treatment can be recommended. No therapy has been shown to be of value in preventing premature births. Symptomatic treatment with sulfonamide drugs and antibiotics is indicated to combat secondary bacterial infection of the genital organs.

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Enzymatic Zonulolysis for Cataract Surgery

Enzymatic zonulolysis, a term recently introduced into the vocabulary of ophthalmic surgeons, refers to dissolution of the zonule of the lens by enzymes. In this report, the enzyme involved was *alpha*-chymotrypsin.

Dissolution of the zonule of the lens facilitates removal. In 22 persons with cataracts, a 1:5,000 solution of enzyme was introduced into the anterior chamber after completion of the corneal section and a peripheral iridotomy. The canula of the irrigating syringe was slipped between the iris and the surface of the lens. Then the enzyme solution was slowly introduced while the tip of the canula was moved in a circular direction parallel to the equator of the lens. The maneuver was completed by irrigating the anterior chamber with normal saline solution to neutralize the enzyme and halt its lytic effect.

A contact time of 2.5 minutes seemed adequate, and dosage of solution seemed to make little difference whether it was 0.5 cc. or as great as 3.0 cc. The lytic effect of this enzyme is sufficiently powerful that overly long periods of contact may cause the lens to drop into the vitreous chamber when the grasping instrument is applied to the capsule.—*Proc., Mayo Clinic, 34, (1959): 478.*

Heredity Influences Mastitis

Mastitis ratings of daughters compared with their dams and mastitis ratings of progeny of different sires show that heredity plays an important part in determining a cow's resistance to mastitis, according to scientists at North Carolina State College.

These workers also found that low udders are more susceptible to mastitis than high udders. This work provides the first known scientific data to support the commonly accepted connection between udder pendulousness and mastitis.—*Breeder's Gaz. (Sept., 1959): 22.*

Fat Necrosis

in Man and Animals

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NECROSIS of fat is a common incidental finding in the course of many necropsies upon domestic animals. It occurs, though apparently less frequently, in man. The lesion is readily recognized by the chalky white, lumpy to granular nodules of altered fat scattered within fat tissue and may occur anywhere in body-fat deposits but is more readily recognized in the fat of the mesentery, omentum, and other extraperitoneal regions. There is seldom any clinical indication of its presence in animals.

In man, the condition may be encountered following localized trauma to fat tissue, as an accompaniment of pancreatitis, as a peculiar palpable nodular lesion in the subcutis of newborn infants (sclerema adiposum neonatorum, scleroderma neonaforum, fat necrosis of newborn), as a complication of hypothermia in infants, in degenerating lipomas, following inadvertent injection of oils into body tissues, and in Weber-Christian disease (relapsing febrile nonsuppurative nodular panniculitis).

In domestic animals, the condition is usually attributed to the liberation of lipase[18] or amylase and lecithinase[19] in pancreatitis, or to pressure and trauma, such as that accompanying parturition or prolonged recumbency. Considering the many times fat necrosis is encountered in the absence of evidence for any of these conditions, it seems probable that at times other factors must be responsible.

It has been suggested that febrile attacks in cattle may result in the alteration of body fat (higher cholesterol content, higher melting point, tendency toward isomerization); the altered fat then acting as a foreign body to induce the characteristic picture of fat necrosis.[13] However, the role of fever remains to be proved. Recently, it has been shown that the feeding of high levels of saturated long-chain fatty acids or their esters (usually glycerides) will produce fat necrosis in rats[6,10,11] and rabbits.[20] Although the levels fed were higher than those encountered in the ordinary diet, the result nevertheless indicates that dietary factors play a role in fat necrosis. Clinical evidence that diet may be important in the development of fat necrosis may be deduced from the statement[15] that cattle in northern China fed high levels of soybean cake develop fat necrosis, whereas those fed only grass do not.

This paper concerns the nutritional aspects of fat necrosis. It will not deal with necrosis associated with pancreatitis or trauma. Neither is it concerned with steatitis, an inflammation occurring in fat apparently because of the presence of peroxides of highly unsaturated fatty acids resulting from vitamin E-deficient diets.

The Pathogenesis of Fat Necrosis

The appearance of necrosis in adipose tissue is quite different from that encountered in most other body tissues. The first detectable changes of fat necrosis are usually the conversion of isolated fat cells from their normal vacuolar condition to one wherein the cytoplasm becomes uniformly a finely granular opaque pink to pale purple color (so-called "saponified fat cell"). The fat then further hydrolyzes to fatty acids and glycerol, the latter of which is soluble, harmless, and is carried away. Unsaturated or short-chain acids which are liquid at the temperature of the surround-

From the Western Regional Research Laboratory, a laboratory of the Western Utilization Research and Development Division, Agricultural Research Service, U.S.D.A., Albany, Calif.

ing tissue dissolve less readily, abstracting basic ions from the surrounding fluids and liberating hydrogen ions. The resultant local acidity produces necrosis.[9,12] The degree of spreading necrosis is somewhat limited, depending upon the length, or (of less significance) the branching, of the acids liberated.[8] Otherwise, the process would become self-perpetuating until all nearby fat was destroyed.

Acute necrosis of nonfatty tissues is usually accompanied by severe vascular and inflammatory cell responses but, because of the compaction of fat cells about vessels and the nature of adipose tissue itself, there is little alteration other than that involving the fat cell. If neutrophils are present, they usually indicate local infection, an extension from inflammation in an adjacent nonfatty tissue, or the presence of some abnormal substance (as in steatitis). Some of the liberated soluble acids may combine with sodium or potassium to form soluble soaps, or with calcium to form insoluble and thus visible soaps.

When longer chain-length acids (higher melting point) are liberated during hydrolysis, they dissolve slowly and act mechanically, in which case the crystals themselves may be visible microscopically. Thus, the picture is less one of necrosis of adjacent cells but rather proliferation of interstitial and macrophage cells, phagocytosis by macrophages, and appearance of giant cells.[9] There is little influx of cells by way vascular or lymphatic channels. The entire process often occurs within the affected fat cells, and physical disruption of the fat cell is not necessary for its occurrence.[23] Affected cells may eventually be replaced by fibrous tissue or may become calcified. More often, they merely remain as seen in the necrotic state. Adjacent fat cells appear perfectly normal. It is possible that other pathways of fat necrosis exist,[22] particularly those induced by diet, but their nature is poorly understood.

The Role of Diet

It has long been known that the feeding of various fats greatly affects the composition of body fat.[7,17] Swine fat normally contains the unsaturated acids oleic, linoleic, and arachidonic, and the saturated acids myristic, palmitic, and stearic. Pigs fed soybeans deposit a fat rich in linolenic acid; those fed peanuts deposit fat with increased content of arachidonic acid.[7] Both are unsaturated acids and tend to produce soft fat.

Pigs fed diets low in fat develop hard fat, up to 97 per cent consisting of the glycerides of stearic, palmitic, and oleic acids. When animals are first starved and then fed various diets, the conversion of body fat is much more rapid, indicating that the turnover of body fat in the normal nonstarved animal is slower than frequently assumed. Thus, rats starved and then fed a diet high in corn oil developed a body fat surprisingly similar in composition to corn oil and containing 16 per cent saturated acids and 84 per cent unsaturated acids. When these animals were again starved then placed on a diet containing sucrose instead of corn oil, the fat became 26.7 per cent saturated and 73.3 per cent unsaturated fatty acids.[17] During starvation, there is no preferential loss of either saturated or unsaturated acids, and the fat chemically resembles that of normal animals.[17]

The occurrence of diets or other factors resulting in deposits of saturated fat predisposes to fat necrosis, or may even produce fat necrosis. Children characteristically have subcutaneous fat higher in saturated fatty acids than do adults.[16,22] One writer[22] describes an infant who had fat with percentages of 65.04 oleic acid (one double bond), 27.81 palmitic, and 3.15 stearic (fat melting point 42 to 43 C.), whereas the acid percentages in fat from a group of adults averaged 86.21 oleic, 7.83 palmitic, and 1.93 stearic (fat melting point 34 to 40 C.). This harder infant fat may result in the spontaneous development of nodular lumps of necrotic subcutaneous fat in the newborn (scleroderma neonatorum). The hard fat of children may also cause the development of such necrotic masses in infants chilled for cardiac surgery.[5]

A similar condition has been reproduced in pigs. One group of young pigs was fed a diet high in soybean oil, another a diet high in hardened (hydrogenated) coconut oil. When these animals were chilled until the body temperature reached 28 C. (82.4 F.) and then warmed, the animals fed soybean oil rapidly returned to normal and had normal fat when biopsied two weeks

later. On the other hand, those pigs fed hardened coconut oil were lethargic and anoretic for several weeks, and their subcutaneous fat developed widespread areas of necrosis.[1] The unsaturated fatty acid content of children's fat increases as they mature, whereas that of swine decreases.[1] The hypothermic changes in children are generally confined to subcutaneous fat.

Since primary necrosis of subcutaneous fat is uncommon in domestic animals, cold is probably not an etiologic factor of concern in them. Interestingly enough, lipoma tissue exhibits a much greater tendency toward fat necrosis than does adjacent normal fat. This may be due to the predilection of lipomas to trauma, but it may also be due to the exceedingly high palmitin content of lipid tumors.[22]

In recognition of the tendency for the body to deposit fat similar to that ingested, it has been proposed that fat necrosis induced solely by the feeding of high levels of hard fat containing long-chain saturated fatty acids may be due to the deposition of some harmful abnormal fat or to the excessive deposition of the ingested saturated fatty acid triglycerides themselves.[6] The deposition of abnormal fat is known to occur since rats fed 18 per cent of partially hydrogenated peanut oil developed high levels of conjugated diene acids (alternating double- and single-bond acids) although the oil fed contained only 0.1 per cent of these.[4] Also, the hydrogenation of vegetable oils induces the formation of unnatural trans acids, one of which (elaidolinolenic), when assimilated, may act as a competitive inhibitor of linoleic lipoxidase.[14] However, it seems likely that the mere feeding of high levels of hard fats results in the deposition of fats containing long-chain saturated acids producing the fat necrosis.[11]

Additional support for this theory is found in the demonstration that the body fat of rats fed high levels of acetylated glycerides of stearic acid would crystallize out of ether solutions at body temperatures, whereas normal body fat would not.[6] This body fat contained no more acetate that did normal fat, and so the peculiarity must be attributed to the stearate moiety. It has been shown that animal's adipose tissue can handle saturated acids in certain ratios to unsaturated acid.[11] When the saturation ratio is exceeded by diet, then crystallization occurs. The effect of this altered ratio may be aggravated by chilling. It has been shown that reversal of the dietary ratio of saturated to unsaturated fatty acids will reverse the necrotic process and healing will occur.[11]

An important factor to consider in the dietary induction of fat necrosis is the digestibility of the various fatty acids fed. Fatty acids having chains of 4 to 10 carbon atoms are 100 per cent digestible;[2,3] above C_{10} digestion decreases with chain length. Stearic acid (C_{18}) is only 12 per cent digestible, and behenic (C_{22}) only 7 per cent. The corresponding mono-unsaturated acids are more digestible than the saturated. Oleic acid (C_{18}) is 84 per cent and erucic (C_{22}) is 53 per cent digestible. This might indicate that the melting point of the fatty acid is a good index of its digestibility, but such is not necessarily the case. Nervonic acid (C_{24}-one double bond) has a melting point of 39 C. and thus would be viscous in the intestine. Stearic acid (C_{18} saturated) has a melting point of 69 C. (156.2 F.) and would occur as a hard solid; yet both have approximately the same coefficient of digestibility. In general, unsaturated fatty acids are better digested as triglycerides, and saturated acids as the free acids. High levels of protein in the diet increase the digestibility of these fats.

An entirely different mechanism for dietary fat necrosis has been suggested.[15] Rabbits fed high levels of raw soybeans, a foodstuff rich in plant lipase, developed high serum lipase levels and when necropsied had necrosis of perirenal fat. Cooking the soybeans prevented both manifestations. The authors concluded that plant lipases were absorbed and responsible for the fat necrosis. However, fat necrosis could not be induced in an experiment involving the feeding of large quantities of soybeans and peanuts to swine.[21] Extensive studies on soybeans the past few years have failed to produce any descriptions of induced fat necrosis. Soybeans and peanuts are both rich in unsaturated fatty acids.

Fat Hydrolysis After Death

Following the death of an animal, the body temperature drops and, concurrently, the fat begins to solidify and whiten in

color. Not infrequently the fat will develop lardaceous, somewhat granular, white nodules which upon microscopic examination are found to contain various degrees of hydrolyzed fat and fatty acid crystals. This probably represents hydrolysis and crystallization of the less soluble fatty acids caused by lowering of the environmental temperature, by the postmortem liberation of lipolytic enzymes, and by lipases from putrefactive organisms. This has not occurred during the life of the animal, hence is not fat necrosis. The nature of this phenomenon may be questioned if some time has elapsed since death or there has been little or no refrigeration of the carcass. Its true nature may be recognized by the primarily visceral location of the process and microscopically by the absence of saponification (soap formation) and the absence of a cellular reaction. In poorly rendered lard, the process may be recognized by smell, by taste, or by the detection of high levels of free fatty acids. It is aptly named "souring."

Summary and Conclusions

Necrosis of fat is commonly encountered during necropsies of domestic animals. It occurs, though apparently less commonly, in man.

The best understood pathway of fat necrosis involves hydrolysis of glycerides to glycerol and fatty acids. Glycerol is soluble and harmless. Fatty acids of low melting point dissolve and may induce local acidity and necrosis. Fatty acids of higher melting point act mechanically and may induce cellular proliferation, phagocytosis by macrophages, and the appearance of giant cells.

Fat necrosis occurs in newborn infants as a consequence of the high level of saturated long-chain fatty acids in the adipose tissue. Such necrosis may occur in children and animals having highly saturated fats if they are exposed to severe chilling.

Fat necrosis may follow trauma, pressure, or may accompany pancreatitis. Recent work has shown that fat necrosis may be induced by diets rich in either long-chain saturated fatty acids or their esters. The role of the diet should be considered in cases of fat necrosis where the causative factor is not obvious.

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Plan Research to Remove Strontium 90 from Milk

A joint research effort to develop dairy-plant methods of removing strontium 90 from milk is being planned by the U. S. Atomic Energy Commission, the U. S. Public Health Service, and the U. S. Department of Agriculture.

Although levels of radioactive fallout from past nuclear testing do not justify action to decontaminate milk supplies, the research is designed to provide practical answers to problems that might arise in the future.

The proposal to develop practical dairy-plant scale methods of removing strontium 90 from milk grew out of laboratory studies which showed that it is possible, on a laboratory scale, to remove strontium 90 from milk through the use of certain chemicals known technically as "ion-exchange resins" which duplicate a natural phenomenon in soils. Strontium 90 that enters the soil is absorbed on soil particles and enters into reactions between the soil and plant root. However, only a little strontium 90 gets into the plant in this way.

It is now known that much of the strontium 90 that does get into plants is absorbed through their leaves. This can be ingested by ruminants.

The best evidence is that the animals take into their systems about 5 per cent of the strontium 90 ingested and that a dairy cow secretes in her milk about 1 per cent of the strontium 90 she consumes each day.

The laboratory tests already done indicate that more than 90 and possibly more than 95 per cent of the strontium 90 that gets into milk can be removed by ion-exchange resins.—*U.S.D.A. News Release, Oct. 5, 1959.*

Efficiency of Feed Utilization in Dairy Cattle

The data from 12 roughage experiments, involving 175 Holstein-Friesian cows, were analyzed to study the variation among cows in their roughage appetite and the efficiency of feed utilization. Highly significant differences were found among cows in their roughage dry matter intake even after adjustment had been made for milk production, body weight, and daily changes in weight. The repeatability of the weekly average of roughage dry matter consumption on a within roughage treatment period and year basis was found to be of the order of 0.70. Cows tended to maintain their rank in roughage comsumption when fed several different kinds of roughage.

The difference among cows in their efficiency of feed utilization was also highly significant. The average efficiency for 20 weeks, starting 60 days after calving, was a requirement of 56.7 lb. of TDN for each 100 lb. of fat-corrected milk produced. Consistent and highly significant differences in efficiency were found in favor of grass silage as a roughage source as compared with hay.—*J. B. Stone et al. Results presented in this paper are from a thesis submitted by J. B. Stone in partial fulfillment of requirements for a Ph.D. degree, Sept., 1959, at Dept. of Animal Husbandry, Cornell University, Ithaca, N.Y.*

Editorial

When the Bombs Fall

Recently distributed was the AMA's "Summary Report on National Emergency Medical Care," prepared at the request of and for the Office of Civil and Defense Mobilization. In this booklet are spelled out the responsibilities and functions of the medical profession when mass casualties occur. How, after adequate preparation and training, health personnel such as veterinarians, dentists, nurses, and many others might support the physician is also suggested in some detail.

Veterinarians in a few cities may already be aware of the role they would be expected to play if mass casualties occur, but most do not. Many will be interested to know that, in the booklet mentioned, it is recommended that veterinarians be trained to perform specific tasks including:

1) First-aid, including but not limited to artificial respiration, emergency treatment of open chest wounds, relief of pain, treatment of shock, and the preparation of casualties for movement.

2) Control of hemorrhage.

3) Attainment and maintenance of patent airway, and intratracheal catheterization, to include tracheotomy.

4) Proper and adequate cleansing and treatment of wounds.

5) Bandaging and splinting.

6) Administration of anesthetics under medical supervision.

7) Assisting in surgical procedures.

8) Insertion of nasogastric tubes to include lavage and gavage, as directed.

9) Administration of whole blood and intravenous solutions, as directed.

10) Administration of parenteral medications, as directed.

11) Catheterization of males and females.

12) Administration of immunization agents, as directed.

13) Sanitation, to include waste disposal; examination of water sources, methods of water treatment and distribution; milk sources, methods of sterilization and distribution; and inspection of foods, to include detection of radioactive contamination.

Plans for mass casualty care, passed down from national origin to local levels for execution, will give veterinarians an opportunity to demonstrate in still another way their willingness to serve their communities as members of health teams. That they will acquit themselves well is to be expected. That their ability to handle casualties will be second only to physicians is also to be expected by us but may surprise others of the health group who have not seen a veterinarian catheterize animals, give parenteral medication, insert stomach tubes, repair fractures, treat wounds, and perform tracheotomy and many other surgical procedures.

It may or may not be safe to presume that those responsible for planning disaster programs are aware that modern veterinary hospitals have x-ray machines, autoclaves, sterilizers, surgical instruments, otoscope-ophthalmoscopes, resuscitators, splints, dressings, bandages, drugs, and many other materials usable in the treatment of man on an out-patient basis.

At any rate, we've not yet seen plans for use of such facilities in event of disaster. Perhaps this will be left to the local director of the program, as will be, no doubt, the selection of which veterinarian in a community will do what. Certainly, the veterinarians actively engaged in practice should be given different assignments from those who may have been engaged in administrative or other nonclinical work for many years.

It should be safe to predict that the physicians responsible for setting up disaster training programs will find veterinarians requiring less training than any other group in their city. The manner in which the veterinarian transfers his medical skills from domestic animals to man, the extent to which he cooperates in these local plans, and the type of relationship established with other members of the health group can do much to further interprofessional and public relations.

from the *Research Journal*

Measuring Growth of Leptospires

Nephelometry was used as a turbidimetric method to estimate the growth of Leptospira cultures. Cultures of *Leptospira pomona* were grown in modified Stuart's medium containing 7 per cent rabbit serum. Three culture units (20 x 150-mm. test tubes, 250-ml. Erlenmeyer flasks, and 300-ml. nephelo-culture flasks) were employed. The nephelo-culture flask consists of an Erlenmeyer flask with a 19 x 130-mm. sidearm cuvette fused to it. Comparison of the different culture units was made by estimating the growth of the *Leptospira* with a Coleman No. 7 nephelometer. This instrument was adjusted to a scale reading of 60 with a biological nephelometric standard. The biological nephelometric standard consisted of *Brucella abortus* 1119-3 USDA tube antigen suspended in 0.5 per cent phenolized saline transmittance at 420 mμ.

The growth data obtained with nephelo-culture flasks was similar to that obtained by more tedious methods. The sidearm flask allows turbidity to be estimated at any time during the growth cycle without removing a sample of the culture. The culture-volume relationship during such estimates remains undisturbed. Little chance of contamination exists. The cell crop can be harvested at terminal incubation with no loss of cells. Inocula can be rapidly, simply, and precisely adjusted. Extensive use has been made of the nephelo-culture flask to study the effect of a wide variety of culture and biochemical conditions upon the growth pattern of Leptospira. Furthermore, it has proven useful in periodically checking the antigen density of cultures used in standardized agglutination-lysis tests.—[*H. C. Ellinghausen, Jr.: Nephelometry and a Nephelo-Culture Flask Used in Measuring Growth of Leptospires. Am. J. Vet. Res., 20, (Nov., 1959): 1072-1076.*]

New Books

A Guide to the Identification of the Genera of Bacteria

In the preface of this book, it is stated that the aim is to place in "the hands of research workers, teachers, and students, a volume in which directives for the identification of bacteria are supported by a complete list of the techniques needed for the purpose." The author has succeeded in preparing a guide which can be quite useful to the experienced bacteriologist and to advanced students. It is intended to be used as a supplement to Bergey's "Manual of Determinative Bacteriology," seventh edition, 1957. Some tests of its use in my laboratory indicate that Bergey's Manual may be easier to use after first consulting this guide.

The first 47 pages are given to a comprehensive key of the genera of bacteria. For each genus, there is given the appro-

priate page number in Bergey's Manual and also references to pages in the guide that have detailed descriptions of the particular genus. In addition, there is alternate terminology as used by Topley and Wilson (4th ed.). A few genera not found in Bergey's Manual are included in the key. The key is largely a dichotomous one. After using the key to determine the genus, one must refer to Bergey's Manual for species identification in most cases.

A section entitled "Digest of Genera" covers 95 pages which contain a great deal of information and differentiating characteristics of each genus. For example, under "Pseudomonas," there is a detailed description of the genus plus some characteristics of certain species. Also some data are recorded, such as: "Indole is produced by 24/125"; "Starch is hydrolyzed by 15/35"; meaning that indole was produced by 24 of 125 species studied, etc. As part of the two-page discussion on "Streptomyces," there is a table listing 101 species and the kind of antibiotic produced by each, if known. There are 31 plates of line drawings or photomicrographs, many of which are subdivided, making a total of 177 illustrations. A section on "Methods" extends for 63 pages. The preparation of mediums, stains, and some kinds of apparatus are well described.—[*A Guide to the Identification of the Genera of Bacteria. By V. B. D. Skerman, 217 pages. Williams & Wilkins Co., Baltimore 2, Md. 1959. Price $5.50.*]—ALFRED G. KARLSON.

Transplantation of Tissues

This second volume is an important addition to the first volume which was published in 1955. Dr. Peer, the editor, and 12 recognized authorities in their field have collaborated to present the latest developments in tissue transplantations. Skin, cornea, fat, nerves, teeth, blood vessels, endocrine glands, organs, peritoneum, and cancer cells are discussed by men who have become specialists in these fields.

The book is primarily a review of the literature as it pertains to each specific type of transplantation. With a few exceptions, very little space is devoted to actual surgical techniques, these being aptly described in other texts. The experimental work on animals and that done on man are discussed separately. The clinical applications resulting from experimental research are noted, making it possible for practitioners to apply them in their surgical cases. There is an excellent discussion on the newer concepts of cells and tissues and a discourse on the behaviour of cells in tissue transplants. Dr. P. B. Medawar has written a chapter on zoologic laws of transplantation which is timely and most provocative.

Each section is well documented as considerable space has been devoted to references. The editor and each author have done an excellent job in presenting the material. The lack of uniformity so frequently observed in a book of multiple authorship has been overcome with pleasing results.

The publisher also deserves recognition for his contribution. The type and the paper are good, making reading easy, and the illustrations (252) are excellent.

Unfortunately for the veterinarian, this book contains few of the many complex surgical procedures which are used in tissue transplantation. Undoubtedly, these might be obtained by checking the references at the end of each chapter. It is realized that, if surgical techniques were included, the size of this volume would be enormous.

For those interested in experimental tissue transplantation, this volume would be an excellent aid to ascertaining the latest developments in this phase of medicine which has gained such significance in our time. To the busy practitioner, especially the physician, this volume affords the latest knowledge on the subject and offers new challenges for its use in practical medicine and surgery.—[*Transplantation of Tissues. Lyndon A. Peer, M.D. (editor) and twelve contributors. Volume II. 690 pages. Williams and Wilkins Co., Baltimore 2, Md. Price $20.00.*]—ELROY C. JENSEN.

News

American Association of Equine Practitioners

The 1959 AAEP convention, held in Chicago, December 14-16, was attended by a record 202 members. During the meeting, more than 80 new-member applications were acted upon favorably.

The following officers were installed: Drs. Jordan Woodcock, Rye, N. Y., president; M. B. Teigland, Opa Locka, Fla., president-elect; F. J. Milne, Toronto, Can., secretary-treasurer; and M. L. Scott, Akron, Ohio, executive secretary. Brigadier General W. O. Kester, Denver, Colo., immediate past-president, was named permanent chairman of the board of directors.

Not only was this convention the biggest in the AAEP's history, but it was characterized by spontaneous audience participation to an extent seldom seen at veterinary meetings. For example, in addition to the seven scheduled speakers on a panel discussion of the 48-hour Medication Rule, more than a score of the conferees spoke from the floor on this subject.

Following the discussion, the Association drafted a proposed substitute for the 48-Hour Rule to be presented to the National Association of State Racing Commissioners in March. The proposal, patterned after the

Some of the first-day participants on the Equine Practitioners' program.

Seated, left to right—Mr. James H. Inglis, president, National Association of Racing Commissioners, Commissioner of Racing—Michigan; Mr. Thaddeus B. Bruno, chairman, Illegal Practices Committee of the National Association of State Racing Commissioners; Mr. Marshall Cassidy, director of racing, New York Racing Association; Dr. Jordan G. Woodcock, Rye, N. Y.

Standing, left to right—Mr. John Manfuso, chairman, Board of Directors of the H.B.P.A.; Dr. William O. Reed, Elmont, L.I., N.Y.; Brig. Gen. Wayne O. Kester, immediate AAEP past-president, Denver, Colo.; Dr. Y. T. Oester (M.D., Ph.D.), professor of pharmacy, Loyola University, Chicago, Ill.; M. B. Teigland, Opa Locka, Fla.

Kentucky rule, provides among other things, that "no internal medication may be administered to a horse the day of its race without specific written permission from the state or official veterinarian after consultation with the stewards."

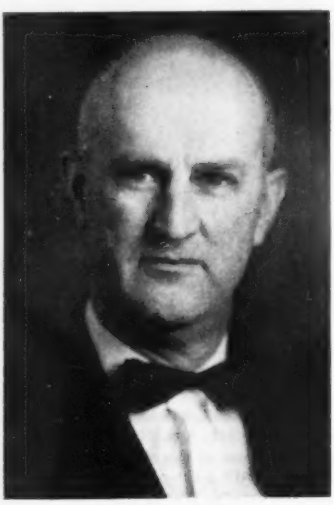
Equine encephalomyelitis also received official attention. In addition to being a program item, it was also the subject of a resolution forwarded to the AVMA. Since this is an avian disease, neither contagious for horses nor transmissible from horse to horse, the Association urged that appropriate steps be taken to re-name it, in order to dissociate the equine species from the disease name.

The resolution also requested dissemination of public information material relative to the disease and indicated that the AAEP recommends vaccination for protection of the individual horse but is opposed to compulsory vaccination which is based on the premise that the horse is a source of infection for man.

Further information on the AAEP convention will be reported in a future issue of the JOURNAL.

Dr. M. R. Clarkson Named Associate Administrator of USDA's Research Service

Dr. M. R. Clarkson has recently been named associate administrator of the U.S.-D.A.'s Agricultural Research Service.



Dr. M. R. Clarkson

As associate administrator, a new position in ARS, Dr. Clarkson shares with Dr. Byron T. Shaw, administrator of ARS, the broad responsibility of coordinating all USDA research as well as administrating ARS regulatory activities.

Born in Ferndale, Wash., Dr. Clarkson received his B.S. degree and D.V.M. degree from Washington State University. In addition, he received an LL.B. degree from Georgetown University.

Dr. Clarkson joined the USDA as a veterinarian in the Meat Inspection Division at Sioux Falls, S.D., in 1930. He was transferred to Washington in 1939, and became assistant chief of the Division in 1944.

In 1947, Dr. Clarkson became chief of the Inspection and Quarantine Division of the former Bureau of Animal Industry and was placed in charge of the program, in cooperation with Mexico, for the control and eradication of foot-and-mouth disease in cattle there. In 1951, he became a special assistant to the administrator and became deputy administrator in 1952.

Dr. W. L. Popham Succeeds Dr. Clarkson as Deputy Director

Dr. William L. Popham, who had been serving as assistant administrator for regulatory programs, has been named to Dr. M. R. Clarkson's former position as deputy administrator.

Agricultural Research Service activities for which Dr. Popham is responsible include: animal disease control and eradication; meat inspection; control and eradication of insect pests and plant diseases; enforcement of plant and animal quarantines and regulatory orders; and the enforcement of other related statutes.

Dr. Popham was born in Cornvallis, Mont., and received his B.S. degree and his honorary D.Sc. degree from Montana State College.

He entered the USDA as a field assistant in the former Bureau of Plant Industry at Bozeman, Mont., in 1922. Shortly afterward, he was placed in charge of state barberry eradication program, and later was made supervisor of barberry eradication field work in Ohio, Indiana, Illinois, Michigan, and Wisconsin.

In 1931, he was named senior pathologist in charge of cooperative federal-state barberry eradication work in 13 North Central

states. Dr. Popham also moved to Washington in 1931. At that time, he was made assistant head of the National Barberry Eradication Program.

He was named assistant chief of the former Bureau of Entomology and Plant Quar-

Dr. William L. Popham

antine in 1941, and became director of the plant pest control division of ARS when it was established in 1953. He has been assistant ARS administrator since 1956.

Brig. Gen. W. O. Kester Named Director of Professional Education by the Morris Animal Foundation

Retired chief of the Air Force Veterinary Service, Brig. Gen. Wayne O. Kester of Denver, Colo., has recently been named director of professional education by the Morris Animal Foundation.

In this newly-created position, he serves as a liaison among the Foundation's board of trustees, the veterinary colleges, and the veterinary profession as a whole.

General Kester served eight years as chief of the Air Force Veterinary Corps and his work with the Morris Animal Foundation closely parallels the veterinary research and training program developed and directed by him in the service.

A prime objective of the Foundation is to encourage and enable students as well as doctors of veterinary medicine to continue in graduate training, teaching, and research.

In accordance with this policy, General Kester is expected to visit many of the colleges, encourage students to become interested in research, and to arrange Foundation grants. He will also interpret and present the findings of these research projects to the veterinary profession.

In recent years, 22 graduates have been assisted by grants from the Foundation, many of whom have attained a Doctorate degree.

This year, there are 15 students enrolled as Morris Animal Foundation Fellows at various veterinary colleges. Most of the research is related to metabolic disease problems in small animal medicine.

Applications for Ralston Purina Research Fellowship Awards Due March 1 for 1960-1961

Application blanks for the Ralston Purina Research Fellowships for 1960-1961 have been mailed to land-grant colleges throughout the United States and to three agricultural colleges in Canada.

Under this program, ten outstanding agricultural college students will be able to do graduate work. Selection of the ten winning students will be made, as in the past, by a committee of five, composed of a representative from the Poultry Science Association, the American Dairy Science Association, the American Society of Animal Production, the American Association of Land-Grant Colleges and State Universities, and the AVMA.

Three Fellowships will be awarded in animal husbandry, three in dairy husbandry, three in poultry husbandry, and one in veterinary science. Each of the Fellowships amounts to $1,800 annually.

The purpose of the Ralston Purina Research Fellowships is to train outstanding college students for leadership in the science of livestock and poultry production. The first Fellowships were granted for the 1949-1950 school term.

This past year, better than 50 per cent of the recipients of the Ralston Purina Undergraduate Scholarships indicated that they will continue into postgraduate work.

This pattern seems to indicate that although undergraduate enrollment has been

declining, a significantly greater percentage of those graduating recognize the importance of advanced work to better meet this challenge.

Application blanks for these fellowship awards may be obtained by writing the Ralston Purina Research Awards Committee, c/o Mr. J. D. Sykes, Ralston Purina Company, St. Louis 2, Mo. Both the application and required information must be in the hands of the awards committee by March 1, 1960.

Canadians Publish Veterinary Journal

On Jan. 1, 1960, the Canadian Veterinary Medical Association published Volume 1, Number 1, of its official monthly—*The Canadian Veterinary Journal.*

It is composed mainly of research papers, practitioners' case reports, and abstracts of new books. The subscription rate is $6.00 per year.

s/Dr. C. D. Maplesden, *Business Manager, Canadian Veterinary Journal.*

Among the States and Provinces

California

San Luis Obispo.—At its regular meeting held on Dec. 3, 1959, the Mid-Coast V.M.A. elected the following slate of officers for 1960: Drs. Jack Sohrbeck, president; Harry Plymale, vice-president; William Matulich, secretary-treasurer.

s/William Matulich, *Secretary-Treasurer.*

Florida

Gainesville, Third Annual Conference.—"Diseases of Young Animals" will be discussed in a symposium at the third annual Florida conference for veterinarians on April 30 and May 1, 1960, at the University of Florida.

Thirty-five subjects will be covered by some of the nation's leading experts on management, nutrition, and diseases of pups, kittens, calves, pigs, and foals.

s/W. R. Pritchard, *Head, Department of Veterinary Science.*

Massachusetts

Boston.—A new record for attendance by New England veterinary medical groups was reached at the four-day combined anniversary celebration of the New England V.M.A. and the Massachusetts V.A., at the Hotel Statler Hilton, Boston, Nov. 7-11, 1959. Registration figures exceeded 500, including veterinarians from the six-state area, their wives, guests, and exhibitors.

The Massachusetts Veterinary Association commemorating its 75th anniversary was host to the New England Veterinary Medical Association marking its 25th anniversary; both groups were joined by members of the American Animal Hospital Association, Region I, on their second annual regional meeting, in Boston.

Representing the AVMA at the combined meeting were: Dr. Samuel Scheidy, president; Dr. H. E. Kingman, Jr., executive secretary; Dr. Donald A. Price, editor-in-chief; Mr. Brian Forster, director, public information; Dr. J. R. Hay, director, professional relations; Dr. Myron C. Fincher, executive board member, District I.; and General J. A. McCallam, Washington representative.

Dr. Frederick G. Ruder, Jr. of Amherst, president of the N.E.V.M.A. and M.V.A., officiated at the meeting.

Michigan

Grand Rapids.—The fifteenth National Conference on Rural Health sponsored by the Council on Rural Health, American Medical Association, will meet at Grand Rapids, at the Pantlind Hotel, Feb. 25-27, 1960.

The theme, "Meeting Challenges with Responsibility," will keynote the Conference. Topics of interest to veterinarians will include: careers in the field of medicine (including veterinary medicine); responsibilities to our older rural population; what we are eating (additives in food); health techniques in 4-H work and immunization for farm families.

s/Drs. J. L. McAuliff and Martin P. Hines, *AVMA Representatives on Advisory Committee to the AMA Council on Rural Health.*

As we went to press, January 20 . . . news reached us of Dr. Erskine V. Morse's appointment as Dean of the School of Veterinary Science and Medicine at Purdue University. A detailed report and a biographical sketch of Dr. Morse's career will follow in our February 15 issue.

Foreign News

Mexico

Mexico 7, D.F.—In the Nov. 1, 1959, issue of the JOURNAL, pp. 497-498, it was erroneously reported that Dr. Alfonso Alexander (MSU '43) was secretary of the Department of Agriculture and Animal Husbandry. His correct title is *advisor to the Department of Animal Husbandry.*

s/Alfonso Alexander, *Correspondent.*

Veterinary Military Service

Canal Zone

U.S. Army Photo.

Fort Clayton—Brig. Gen. Russell McNellis, center, chief of the veterinary division, confers with Col. Homan E. Leech, left, U.S. Army Caribbean surgeon, and Lt. Col. George D. Batcheldor, right, chief of the command's veterinary division.

General McNellis was in the Canal Zone recently to observe and inspect veterinary activities performed at USARCARIB posts and at the U.S. Army military mission in Panama.

Deaths

★**Joseph D. Cozzens** (OSU '25), 63, Big Bear Lake, Calif., died while on a hunting trip in Mound City, S. D., Oct. 16, 1959. Cause of death was attributed to embolism of the pulmonary artery.

Dr. Cozzens moved to Southern California in 1926, largely conducting his practice in Santa Monica and West Los Angeles.

Due to ill health, he retired from practice in 1950. Dr. and Mrs. Cozzens spent much time traveling in Europe and the United States in recent years.

Walter Jonas Hadder (GR '09), 83, Mecosta, Mich., died in October, 1959.

Dr. Hadder had retired from practice five years ago.

George L. Helterline (KCV '15), 70, Eugene, Ore., died in Portland, Ore., on June 7, 1959, after a long illness. He had suffered from lateral sclerosis.

Locating in Eugene in 1922, Dr. Helterline gave up his practice there to become a full-time meat and dairy inspector for the city. He retired in 1949.

Dr. Helterline was secretary of the Oregon V.M.A. from 1925 to 1945.

Nicholas Andrew Panayotoff (OSU '21), 74, Akron, Ohio, died Nov. 5, 1959, after a long illness.

A native of Bulgaria, he came to the United States in 1905, moving to Akron in 1922, from Columbus. He had never engaged in practice.

James C. Powell, Jr. (API '46), 37, Savannah, Ga., died Sept. 10, 1959.

Dr. Powell had been an invalid since being stricken with polio five years ago.

Clayton Witty (ONT '12), 68, Elmore, Ohio, died in a Toledo hospital on Sept. 4, 1959, following an operation for cancer.

Dr. Witty had practiced until ten days prior to his death. From 1928 to 1932, he had served as a member of the Elmore village council and from 1932 to 1940, he was mayor of Elmore.

Women's Auxiliary . . .

Eighth Edition of Auxiliary News Due Off the Press this Spring

BIG SPRING, TEXAS.—The first edition of the AVMA *Auxiliary News*, brain-child of Mrs. H. S. McDonald, Auxiliary president, and Mrs. Russell A. Runnells, then president-elect, was printed in 1953.

At that time, approximately 3,000 copies were distributed to Auxiliary and Student Auxiliary members. This spring, with the publication of the eighth issue, the AVMA *Auxiliary News* will reach almost 9,000 women.

In 1954, publication of the News was turned over to Mrs. H. S. Atkins and, in 1958, Mrs. W. H. Riser became the editor. Interestingly enough, the growing strength of the Auxiliary is reflected in the yearly expansion of the News' mailing lists.

Since the *Auxiliary News* is the only personal contact many of the state and provincial auxiliaries have with each other, it becomes increasingly important to include news from each and every one.

Student Auxiliaries Included

For the first time this spring, the News will incorporate timely topics from nineteen of the Student Auxiliaries. Having their own Honor Roll this year, the Student Auxiliaries will be given credit for reports of their meetings—just as the state and provincial auxiliaries currently do.

Either unusual projects or outstanding members, or both, will be featured in this 1960 edition. And, as always, pictures will be welcomed. (A check for $4.50 should be included with each picture to cover the cost of making a cut.) Deadline for news articles will be Feb. 15, 1960.

Convention Coverage

Since the AVMA's Ninety-Seventh Annual Meeting will be held in Colorado, the convention city, "Mile High Denver," and its points of interest will also be given special treatment in the spring issue of *Auxiliary News*. An outline of entertainment for the entire family is being planned to help make this Convention, one of the best.

s/MRS. AKIN M. SIMPSON,
Vice-President for Publications.

Idaho

TWIN FALLS.—The Women's Auxiliary to the Idaho V.M.A. met at the same time as the State Association at the Rogerson Hotel in Twin Falls, at separate sessions, Oct. 10, 1959.

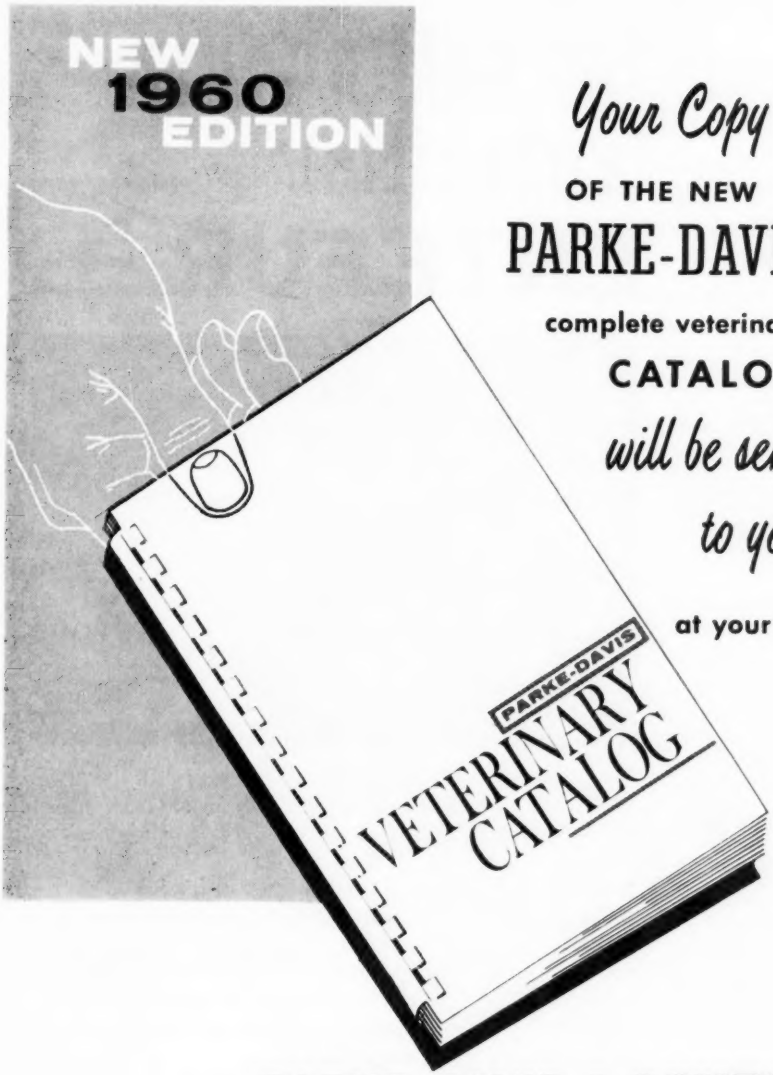
Mrs. J. W. Bailey, Moscow, reported some of the highlights of the AVMA's Ninety-Sixth Annual Convention in Kansas City, Mo.

During the business meeting, plans were sketched to increase the Auxiliary's membership. Particular attention was given to that fact that the Idaho Auxiliary was formed in 1946, with 22 charter members. Preliminary plans were made for the annual state convention scheduled for sometime in June at the Shore Lodge in McCall.

Mrs. H. W. Ronk, Twin Falls, was assisted by Mrs. I. M. Jackson, Kimberly, and Mrs. D. A. Jackson, Twin Falls, in making the arrangements for the luncheon and meeting.

Mrs. Millard W. Ickes, Nampa, was chosen to receive a fall cornucopia.

s/MRS. H. W. RONK, Secretary.



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bactericidal action promptly controls symptoms in 93-95%[1,2] of calves

no development of resistant bacterial strains or of cross-resistance

Remissions of calf scours following treatment with ENTEFUR have been of a dramatic nature and uniformly high. In widespread field tests, cure rates of 93% and 95% have been obtained.[1,2]

ENTEFUR is a bolus containing the new antibacterial nitrofuran, FURAMAZONE® (brand of nifuraldezone) 1 Gm., and bismuth subsalicylate 0.26 Gm. for its mildly astringent, antidiarrheal action. FURAMAZONE is a *new* nitrofuran selected for its specific bactericidal activity against gram-negative and gram-positive enteric bacteria, including the virulent strains of E. coli found in calf enteritis.

ENTEFUR is nontoxic even when administered in doses well above therapeutic levels.

Dosage: 1 bolus perorally, twice daily, for 2 or 3 days.

Supplied: Boluses of 3.3 Gm. each, in box of 24.

1. Osborne, J. C. *in* New Horizons in Chemotherapy. Proceedings of First Regional Conference on the Nitrofurans in Veterinary Medicine. In press.
2. Bull, W. S.: N. Amer. Vet. 38: 3 (Jan.) 1957.

Available through your professional veterinary distributor

NITROFURANS—a *new* class of antimicrobials—neither antibiotics nor sulfonamides

EATON LABORATORIES, NORWICH, NEW YORK

WHAT IS YOUR *Diagnosis?*

Make your diagnosis from the picture below—then turn the page ▶

Fig. 1—Radiograph, ventrodorsal view, taken before injection of radiopaque medium.

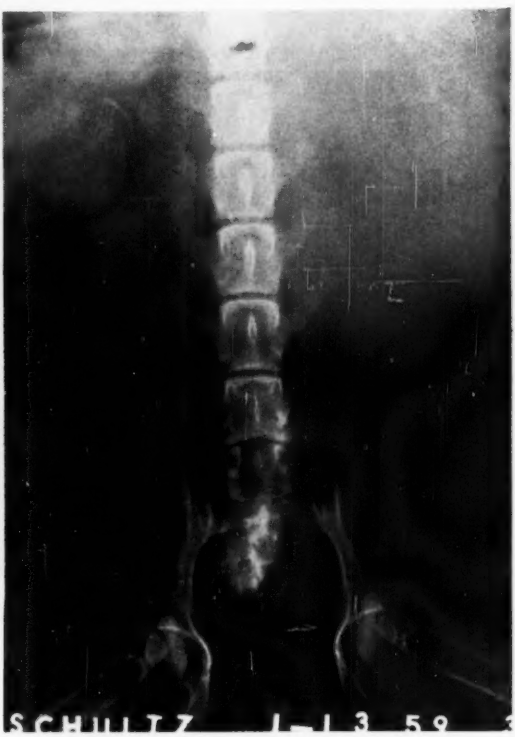

Fig. 2—Radiograph, ventrodorsal view, taken 15 minutes after intravenous injection of radiopaque medium.

History.—A spayed female Cocker Spaniel, 3 years old, had become sluggish and seemed to tire easily. She was normal in other respects including appearance and appetite. In the left lumbar area of the abdominal cavity was a movable, smooth, oval-shaped mass estimated to be about 15 cm. in diameter. The dog had been spayed at 1½ years of age but otherwise had not been ill. Radiographs (fig. 1 and 2), ventrodorsal views, were taken. Figure 2 was taken 15 minutes after an iodine preparation* (50%) was given intravenously.

*Hypaque, a product of Winthrop Laboratories.

Here Is the Diagnosis

(*Continued from preceding page*)

Diagnosis.—Hydronephrosis of the left kidney.

Comment.—The hydronephrosis was the result of a ligature that obstructed the left ureter. The suture was evidently placed there when the dog was spayed.

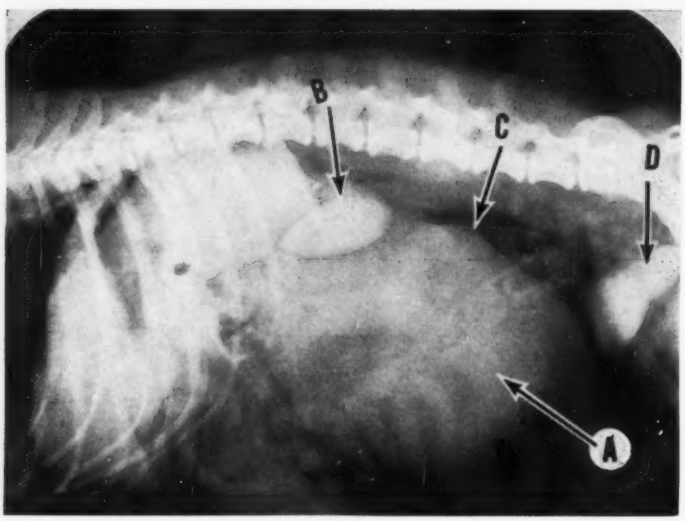

Fig. 3—Radiograph, lateral view, showing cystic kidney (A), right kidney (B), right ureter (C), and bladder (D), outlined by intravenous Hypaque.

Recovery was uncomplicated following removal of the cystic left kidney. Notice that in figure 3 the radiopaque medium is visible in the cystic kidney (A), in the right kidney (B), right ureter (C), and bladder (D).

This report was submitted by William I. Gay, D.V.M., Bethesda, Md., and prepared with the assistance of Dr. Wayne H. Riser, D.V.M., M. S. Kensington, Md.

Our readers are invited to submit histories, radiographs, and diagnoses of interesting cases which are suitable for publication.

for retained placenta

FUREA® *bolus veterinary*

FUREA effectively combats the dangers that arise in bovine retained placenta by a 2-way action:
1) *bactericidal*, even in the presence of pus, blood and serum, and 2) *proteolytic* for resolving necrotic tissue.
Clinical usage produced a rapid decrease in signs of infection, reduction or elimination of malodor, and unimpaired fertility in over 90% of treated cows.
FUREA is an intrauterine bolus containing FURACIN® (brand of nitrofurazone) 0.12 Gm. and urea 12 Gm. The bolus disintegrates swiftly in uterine fluids, providing rapid and uniform distribution of active ingredients to the affected tissues. It is nonirritating and nontoxic.
Dosage: Two boluses inserted into the recently pregnant horn. One bolus may be placed in the nonpregnant horn. Repeat at 24-hour intervals, as necessary.
Supplied: Bottle of 25 boluses.

1. Jones, S. V.; Belloff, G. B., and Roberts, H. D. B.: Vet. Med. 51:413 (Sept.) 1956.

prevention of uterine infection— early restoration of fertility in over 90% of treated cows[1]

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NITROFURANS—a *new* class of antimicrobials—
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History of AVMA

1879

At the meetings for 1879, held again in Boston and at the American Veterinary College, four new members were admitted, including W. B. E. Miller, who was later president of the Association.

Six hundred copies of a petition concerning contagious diseases of domestic animals were printed and sent to members of Congress, to the heads of departments of the government, and to members of the Association.

Papers were presented on "Contagious Pleuro-Pneumonia," by J. D. Hopkins; "Rupture of the Flexor Metatarsi," by A. Liautard; and on the recurring theme of "Cerebro Spinal Meningitis," by I. Michener. Elected as the new slate of officers were: president, J. L. Robertson; vice president, J. H. Stickney; secretary, A. A. Holcombe; and treasurer, Charles Burden.

A USVMA petition was drafted calling for a national ". . . Veterinary Sanitary Bureau, whose duty it shall be to advise Congress as to what measures shall be necessary to control, restrict, or eradicate any contagious or infectious disease affecting the domesticated animals." Specifically mentioned were lung plague and Texas fever, for which "Congress is . . . requested to appropriate a sufficient sum of money to enable the Veterinary Sanitary organization to deal at once and effectually with these important matters. When legislation to this end was introduced, it met initial defeat at the hands of Texas legislators, who were successful—temporarily—in legislating Texas fever out of existence.

In noting the action of the Association to petition for establishment of the Bureau, the *Medical Record* had observed, ". . . .the present status of Veterinary Medicine is so undefined that a National Bureau would not have much more of legitimate professional basis than a National Bureau of Barbers to keep themselves informed upon sycosis."

This brought a wrathful reply from "A Veterinarian" (Liautard?), who observed (among other comments in stronger language): ". . . . the virtue of the medical profession is not entirely above reproach." Although the editor of the *Record* was perhaps less a spokesman for the medical profession than Liautard was for the veterinary profession, such backbiting was not in good taste.

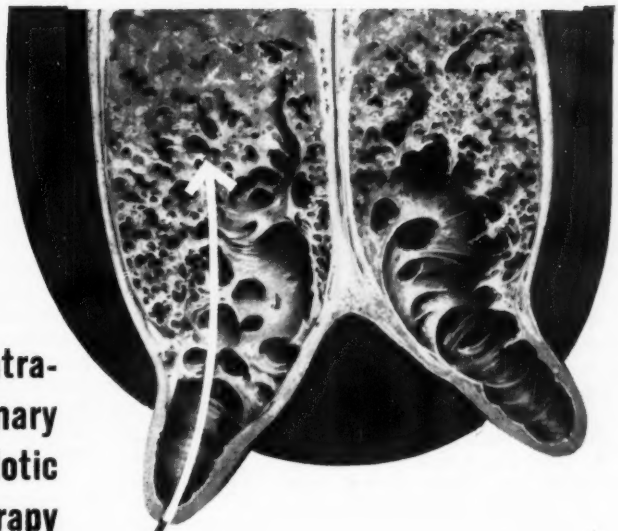
At this time, and for a number of years following, the AVMA membership constituted only about ten per cent of the profession and attendance at meetings rarely exceeded 25 or 30 persons—fewer than present at the original meeting in 1863. Records indicate that no more than a hundred men had been admitted to membership, including the founding forty. The years had begun to take their toll both by death and delinquency leaving the Association only about 65 members.

J. L. Robertson, tenth president of the U.S.V.M.A., 1879-1881.

✦ ✦ ✦

JAMES LINDSAY ROBERTSON, V.S., M.D., D.V.S., tenth President of the USVMA, was born about 1842. He was graduated from the New York College of Veterinary Surgeons in 1867 and the medical department of the University of the City of New York in 1869. After interning, he joined the faculty of the NYCVS and in 1876 secured a second veterinary degree from the American Veterinary College. He then became professor of theory and practice at AVC, later at the combined NY-AVC. Dr. Robertson was retired as Professor Emeritus after 40 years of teaching and practice. He died in November, 1912.

Dr. Robertson joined the USVMA in 1868, and served as its secretary 1869-1874, and as president 1879-1881. Dr. Liautard called him "my boy," and of him it was said: "His thorough knowledge of his subject and his kindly nature won the hearts of all whose privilege it was to have been his students."



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Liquamast is completely soluble in milk, thereby providing maximum antibiotic concentrations therein.

Liquamast diffuses readily, via the milk, to the sites of infection in the upper portions of the mammary gland, whereas oil or ointment type vehicles have a tendency to settle in the main duct of the udder. In extremely deep-seated, chronic cases, Liquamast is a good adjunct to systemic oxytetracycline therapy.

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Committee Reviews 1959 Farm-City Week

The 1959 Farm-City Week, held November 23-29, was termed the most successful in five years by the National Farm-City Committee which met in Chicago December 17. Since 1954, Thanksgiving week has been designated "Farm-City Week" for the purpose of bringing about a better understanding between rural and urban people and to recognize the efforts of individuals, organizations, and communities who work toward this goal.

Approximately 9,000 United States and Canadian communities participated in the observance. So extensive was the publicity that practically every man, woman and child in English- and French-speaking North America read about or heard about the 1959 Farm-City Week.

Informal get-acquainted meetings and panel discussions of mutual problems as farm marketing, retail food prices, high cost of urban labor, and part-time job opportunities for farmers helped to bring the farm and city closer together. The Farm-City Committee cited an instance in which one farm organization invited a group of businessmen to participate in a farm breakfast and morning chores; in another town, a bank displayed a live pig with a chart comparing the cost of raising the pig with the market price; the Chicago VMA and the Northern Illinois VMA instituted the St. Charles Clinic to help urban and rural veterinarians understand each other's problems, and to permit both groups to benefit from methods and surgical techniques which are used in both types of practices.

In planning for Farm-City Week, 1960, the committee named as chairman Robert D. McMillen, public relations director for Corn Industries Research Foundation, Washington, D. C. Co-vice chairmen are C. Maurice Wieting, Ohio Farm Bureau Federation, and Howard C. Tuttle, public relations manager of the tractor and implement division of the Ford Motor Co., Birmingham, Mich. L. A. Hapgood, director of program development, Kiwanis International, Chicago, was named secretary for the fifth consecutive year, and Kenneth Anderson, National Committee on Boys' and Girls' Work, 4-H Clubs of America, Chicago, was named treasurer for the second year.

The Farm-City Committee meeting, Seated at the head table (left to right)—Mr. L. A. Hapgood, Mr. Roy Battles, Mr. Robert D. McMillen, Dr. C. Maurice Wieting, and Mr. Kenneth Anderson.

First row (left, nearest camera)—Mr. Percy Shue, Mr. Herbert Bain, Mr. Earl D. Merrill, Mr. Paul B. Ostendorf, Mr. Walter B. Garver, Mr. Harry Bryson, Mr. Elmaar H. Bakken, Mr. Howard McClarren, Mr. Merle H. Tucker.

Second row (center, nearest camera)—Mrs. Edith T. Bennett, Mr. Brian M. Forster, Mr. John L. McGehee, Mr. Russell E. Frost, Mr. Hugh Muncy, Mr. Dave O. Thompson, Sr., Mr. Frank M. Atchley.

Third row (right, nearest camera)—Mr. Carl E. Rose, Mr. R. H. McDougall, Mrs. Ruth H. Lane, Mr. C. H. Fields, Mrs. Viola Armstrong, Mr. Glenn W. Sample, Mr. C. Dana Bennett.

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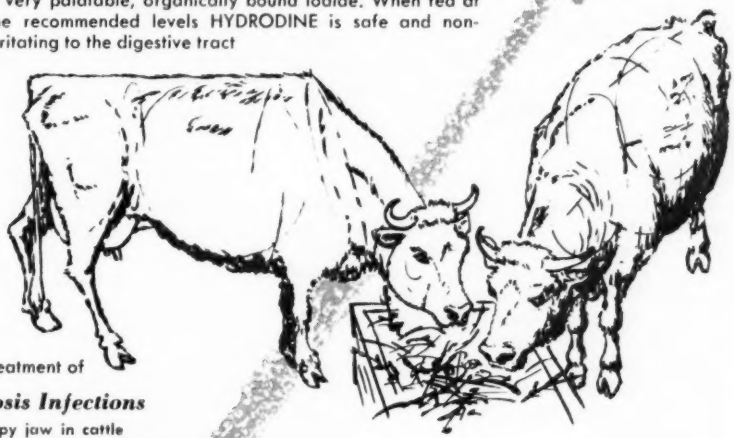
a very palatable, organically bound iodide. When fed at the recommended levels HYDRODINE is safe and non-irritating to the digestive tract



A specific for the treatment of

- ***Actinomycosis Infections***
  Lumpy jaw in cattle
  Fistulous withers in horses
- ***Actinobacillosis***
- ***Infectious Pododermatitis***
  (Foot Rot)
- ***Thyroid dysfunction***
- ***Chronic coughing***

also as an aid in the correction of sterility, chronic udder infections and chronic iodine deficiencies. When fed at low levels Hydrodine will help prevent the occurrence of foot rot.

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An Economist Defends Drug Costs

"If drug prices had risen as much as other prices since 1939, it would cost the consumer at least an additional billion dollars to buy the drug preparations now consumed."

This statement, relayed by the Health News Institute, was made in a speech by Professor Jules Backman, New York University economist, before the American Pharmaceutical Association.

Prof. Backman takes issue with those critics of the pharmaceutical industry who claim that drug prices are rising too high. Americans today spend only 0.1 per cent more of their after-tax income on drugs and medicines than they did twenty years ago, said Prof. Backman.

In 1939, nine tenths of a cent of each dollar left after taxes was spent on medication. Today, despite the post-war inflationary spiral, only one penny of each dollar of after-tax income goes for medication. During this same period the percentage of the medical care dollar spent on drugs and medicines actually *decreased* by 7.5 per cent, despite the "major developments in the form of so-called miracle drugs," said Dr. Backman.

In addition, Americans spend less per person on drugs and medicines ($18.75) than they do for tobacco ($36.00), alcoholic beverages ($53.00), and personal care such as beauty shops, barber shops and toilet articles ($24.00).

Why, then, such widespread criticism of drug prices as being too high? Professor Backman believes that it comes about because expenditures for drugs are made in a short period of time, unlike other purchases which are spread throughout the year. As a result, he feels there is a greater tendency to focus attention on concentrated expenditures for medical care.

However, the economist reminds us that when we pay for our prescription, we are really paying for four things: (1) the product we need, when and where we need it, (2) the professional skill of the pharmacist, (3) the best product for the specific health problem from a research-minded industry, and (4) a share in the research which tomorrow, or next year, or ten years from now will surely discover the answers to the great killers and disablers which still afflict mankind.—*Health News Inst., Oct. 6, 1959.*

An excellent opportunity in the commercial field

Long-established producer of ethical biologicals and pharmaceuticals offers an unusual career opportunity in the sales department of its Home Office. The opening is one for an ambitious, creative young man to administer and originate merchandising plans. A veterinarian, 25-35, who is presently successful in practice or in the commercial field is preferred, although applications are also invited from those without a veterinary medical degree, but with medical sales or advertising experience. Sales aptitude and writing ability are requisites, of course. Send complete resume of experience and qualifications, together with salary requirements, in confidence, to

Box B 17, Journal of the A.V.M.A.

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ULTRACORTENOL®

(prednisolone trimethylacetate CIBA)

HELPS RESTORE MILK PRODUCTION WITH A SINGLE INTRAMUSCULAR DOSE

ULTRACORTENOL is the new and improved analog of prednisolone. It is ". . . the most active glucocorticoid in common veterinary use . . ."[1] Ultracortenol differs from other available corticosteroids in that a single injection provides *optimal duration of activity:*

1. Action is 2 to 3 times longer—sufficiently sustained to minimize the likelihood of relapses.
2. Action is not so prolonged that lactation is unduly suppressed.
3. Total therapeutic activity is greater.

EFFECTS OF ULTRACORTENOL* IN RESPONSIVE COWS[1,2]	TIME REQUIRED
Clinical improvement	12 to 24 hours[1]
Marked increase in milk production	after 24 to 48 hours[1,2]
Appetite restored and maintained, depression disappears, odor of acetone hardly noticeable on breath and in milk	48 to 96 hours[1]

**Ultracortenol dosage may be reduced 50 per cent by administering simultaneously with intravenous glucose.*

SUPPLIED: MULTIPLE-DOSE VIALS, 10 ml., each ml. containing 10 mg. or 25 mg. prednisolone trimethylacetate in suspension for injection. ULTRACORTENOL is available from ethical veterinary distributors throughout the United States.

REFERENCES: 1. Vigue, R. F.: J.A.V.M.A. *133*:326 (Sept. 15) 1958. 2. Vigue, R. F., Fitzgerald, W. H., and Castrucci, R. F.: Vet.Med. *54*:129 (March) 1959.

C I B A
SUMMIT, N. J.

EFFECTIVE VETERINARY DRUGS, PRODUCTS OF EXACTING RESEARCH

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SULFA

...for a new

—one dose produces therapeutic blood levels lasting up to 48 hours in cattle

More than any other professional man, the practicing veterinarian knows that his most valuable asset is time. In any area of his practice, the success of his therapy cannot be measured by results alone; he is a busy man whose time must be used judiciously.

Well aware of this problem, Merck research workers have designed an antibacterial agent which, from its conception, was meant to satisfy the particular needs of the veterinarian. In cattle, this new drug produces effective therapeutic blood levels for at least two days, freeing the veterinarian from time-consuming return calls and minimizing stress-producing handling.

The most exciting development in sulfa therapy in 20 years

SULFABROM gives you all the benefits of sulfa efficacy and at the same time helps eliminate the necessity for frequent administration. Thus, SULFABROM is economical—your initial expenditure is sulfa-low and decreased total dosage brings cost down even lower.

SULFABROM, administered orally or

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BROM*

concept in sulfonamide therapy

SULFABROM is a bromine-substituted sulfamethazine developed by Merck research workers. This new antibacterial agent is the only sulfa product available *exclusively* to veterinarians.

intraperitoneally to cattle, produces effective blood levels lasting up to 48 hours—frequently long enough to eliminate *any* repetition of dosage.

SULFABROM—quickly absorbed, slowly excreted

Although SULFABROM is notable for producing effective levels in rapid time, once it has entered the blood stream its speed of action slows down considerably. SULFABROM is excreted very slowly; this accounts for its long-lasting effect. In cattle, detectable amounts may be present in the urine for as long as six days. Blood levels remain high, sometimes for as long as 53 to 60 hours. And, because it is excreted so slowly, seldom is the amount of SULFABROM passing through the urinary tract ever large enough to cause crystalluria.

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By maintaining a high sulfonamide level in the tissues, SULFABROM minimizes the emergence of resistant strains of most pathogens. SULFABROM exerts its antibacterial effect at the cellular level long enough to combat effectively both gram-negative and gram-positive organisms including those responsible for:

calf diphtheria "recovery in 48 hours";
acute septic mastitis "uneventful recovery";
metritis "back on feed and eating normally" in two days;
foot rot "in 48 hours the cow was able to stand and started to eat";
pneumonia "in five days' time this calf was back to normal";[1]
as well as *scours*, winter dysentery, coccidiosis, shipping fever, listerellosis and miscellaneous infectious conditions, such as peritonitis and infected wounds.

SULFABROM—single-dose sulfa therapy available exclusively to veterinarians

In sum, SULFABROM represents the very latest advance in sulfa therapy. Effective against a full range of infectious diseases, economical to use by any standards, SULFABROM is your answer to the pressing problem of repeat calls and handling time in the treatment of almost any infection.

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DOSAGE *(in cattle)*

SULFABROM Boluses
60-90 mg. (1.0-1.5 grains)/lb. of body weight, orally—for *sustained* levels

SULFABROM Buffered Powder
15-30 mg. (0.25-0.5 grains)/lb., intravenously—for *immediate* levels
30-60 mg. (0.5-1.0 grains)/lb., intraperitoneally—for *rapid, sustained* levels
60-90 mg. (1.0-1.5 grains)/lb., orally, as a drench—for *convenient* levels

Supplied as
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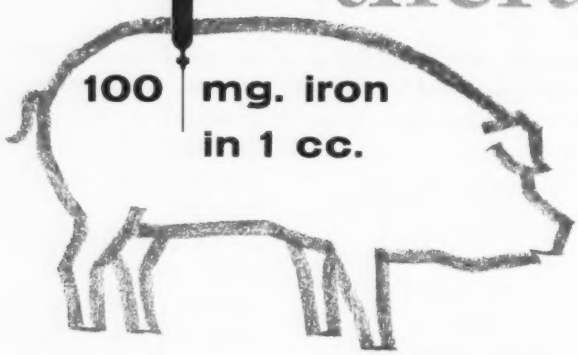

In iron-deficiency anemia of pigs, injectable iron is the therapeutic agent of choice. There are four major reasons why PHARMATINIC 100 should be *your* iron injectable of choice.

Prevention and Treatment in One Injection—PHARMATINIC 100 has a twofold action resulting first in an immediate release of iron to provide prompt hemoglobin response and a secondary release which provides for maintenance of hemoglobin levels until creep feeding has had its effect.

Easier Administration—The high ratio of iron to polysaccharide in PHARMATINIC 100 results in lower viscosity. Pigs experience less pain and soreness—quickly return to suckling after injection. Thus, fatal hypoglycemias are not precipitated.

Economy of Time and Money—Both you and the owner save because PHARMATINIC 100 provides the necessary 100 mg. of elemental iron in a single 1-cc. injection.

Safety—No toxicity or shock has been reported following intramuscular injection of PHARMATINIC 100—even for pigs injected within 24 hours of birth.

PHARMATINIC 100 (I.M. use only) is available in vials of 20, 50, and 100 doses, each of 1 cc. Each 1-cc. dose contains the equivalent of 100 mg. elemental iron in complex with a dextran derivative.

PHARMATINIC* 100

Injectable Iron-Dextran Complex, Pharmachem Specialties, Inc.

SOLD ONLY TO VETERINARIANS

Inquiries from veterinarians and wholesalers are invited.

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is still the soundest approach to the CANINE DISTEMPER "complex"

Throughout the world, Virogen . . . the first multivalent vaccine developed for protection against the "complex" of canine distemper . . . has established an unsurpassed record of safety and effectiveness. For more than 25 years it has often been imitated, but never duplicated.

When the growing threat of infectious canine hepatitis led to the introduction of Virogen D-H, in 1953, veterinarians quickly endorsed this logical expansion of "the Virogen concept."

Today, the increasing incidence of canine leptospirosis shows the clinical need for a single vaccine that will protect against all three of the major viral infections in dogs . . . as well as the secondary bacterial invaders that may complicate an attack of distemper, hepatitis, or leptospirosis.

Such a vaccine, in keeping with the original Virogen concept of "the greatest possible protection," is Virogen D-H-L.

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TERRAMIX® A-B-D-25

oxytetracycline with vitamins

broad-spectrum antibiotic therapy
PLUS *four essential vitamins*

each lb. contains:
25 Gm. oxytetracycline HCl activity
2,000,000 units vitamin A
200,000 units vitamin D_3
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oxytetracycline with glucosamine and vitamins

FORTIFIED SOLUBLE POWDER

broad-spectrum antibiotic therapy, with glucosamine,
PLUS *eight vitamins*

each lb. contains:
25 Gm. oxytetracycline HCl
500,000 I.U. vitamin A
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200 mg. vitamin K
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Available in 1/4 and 1/2 lb. bottles and 5 lb. canisters.
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Joint Committee on Education

At a meeting on Dec. 8, 1959, at the AVMA office in Chicago, the Joint Committee on Education (representing the AVMA Council on Education, the Association of Deans, the Veterinary Section of the Assn. of Land Grant Colleges, and the AVMA Executive Board) studied the status of several bills introduced in the 86th Congress pertaining to federal aid for medical, dental, and public health educational facilities.

The group also considered future needs in veterinary education and recommended that proposed legislation be drafted providing for federal appropriations, on a matching basis, of $5,000,000 a year for 5 years for the expansion of existing schools and $1,000,000 a year for 10 years to construct schools.

Members of the Joint Committee on Veterinary Education: Dr. H. E. Kingman, Jr., Chicago, secretary; Gen. J. A. McCallam, Washington, Dr. D. J. Anderson, Fort Worth, Texas, Dr. G. C. Holm, Stillwater, Okla., and Dr. J. E. Greene, Auburn, Ala.

Nutrition Research Reported at Purdue Swine Day

Two single antibiotics, erythromycin and spiramycin, and two combinations, penicillin and streptomycin, and penicillin, streptomycin, and sulfaquinoxaline increased daily gains in swine 26 to 27 per cent.

A combination of chlortetracycline, penicillin, oxytetracycline, and zinc bacitracin increased gains 21 per cent. A combination of oleandomycin and oxytetracycline increased gains 19 per cent.

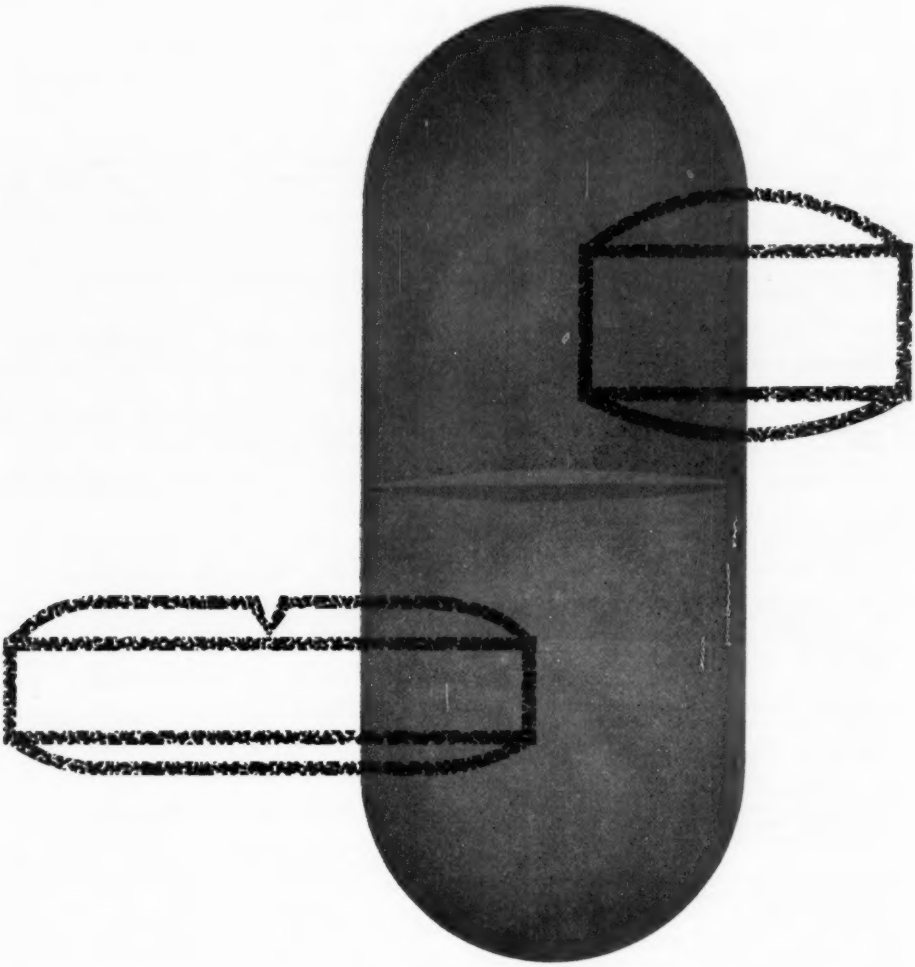
Arsanilic acid increased gains 18 per cent.

No feed additive resulted in a marked improvement in feed efficiency. However, arsanilic acid improved feed efficiency more than any of the additives tested—an improvement of 2.8 per cent over the controls.

A study testing the effects of three different enzyme preparations added to rations for growing-finishing hogs was reported. Pigs fed enzymes gained—1 to 7 per cent less than control pigs. Feed costs per hundred pounds of gain reflected a slight improvement in feed efficiency, but costs of the enzymes were not included.

A study on tranquilizers fed to growing hogs in a previous project indicated that tranquilizers had no significant effect on daily gain or feed consumption.

In the latter experiment, higher levels of tranquilizers were fed. Results of the two trials showed that addition of tranquilizers failed to increase daily rate of gain, stimulate feed consumption, or improve feed efficiency.—*Nat. Hog Farm. (Oct., 1959): 11.*



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COMING MEETINGS

Notices of coming meetings must be received 30 days before date of publication.

Oklahoma Veterinary Medical Association. Forty-fifth annual meeting. Biltmore Hotel, Oklahoma City, Feb. 1-2, 1960. W. D. Speer, 923 E. 6th St., Tulsa, Okla., secretary.

California Veterinary Medical Association. Midwinter conference. University of California, Davis, Feb. 1-3, 1960. Mr. Kenneth Humphreys, 3004 16th St., San Francisco 3, Calif., executive secretary.

California, University of. Western Poultry Disease Conference. University of California, Haring Hall, Davis, Feb. 1, 1960. L. G. Raggi, chairman.

Nebraska, University of. Conference on Swine Repopulation. University of Nebraska, College of Agriculture, Agricultural Experiment Station, Lincoln 3, Feb. 3-5, 1960. Inquiries should be directed to: Dr. E. Crosby Howe, Dept. of Animal Pathology and Hygiene, University of Nebraska, Lincoln 3.

American Animal Hospital Association, Region 2. Annual meeting. Robert Meyer Hotel, Jacksonville, Fla., Feb. 6-8, 1960. R. P. Knowles, 2101 N.W. 25th Ave., Miami 42, Fla., chairman.

Colorado State University. Twenty-first annual conference for veterinarians. Glover Veterinary Hospital, College of Veterinary Medicine, Colorado State University, Fort Collins, Feb. 15-17, 1960. O. R. Adams, program chairman.

Illinois State Veterinary Medical Association. Seventy-eighth annual convention. Abraham Lincoln Hotel, Springfield, Feb. 15-17, 1960. C. B. Hostetler, 1385 Whitcomb Ave., Des Plaines, Ill., executive secretary.

Missouri Veterinary Medical Association. Sixty-eighth annual meeting. Hotel President, Kansas City, Mo., Feb. 21-23, 1960. Paul L. Spencer, P.O. Box 283, Jefferson City, Mo., secretary-treasurer.

West Virginia Veterinary Medical Association. Winter meeting. Greenbriar Hotel, White Sulphur Springs, Feb. 28-29, 1960. Harry J. Fallon, 200 Fifth St., West Huntington, W. Va., secretary.

Indiana Veterinary Medical Association and Vita Vet Laboratories. Third symposium on therapeutic nutrition. State Board of Health Building, Indianapolis, March 4, 1960. E. E. Slatter, Box 108, Marion, Ind., chairman.

Foreign Meetings

International Association of Veterinary Food Hygenists. Second Symposium. Basel, Switzerland, May 15-21, 1960. Dr. A. Clarenburg, 1, Sterrenbos, Utrecht, The Netherlands, president.

Fourth International Congress on Animal Reproduction. The Hague, Netherlands, June 5-9, 1961. For additional information contact: the Secretariat of the Fourth International Congress on Animal Reproduction, 14, Burgemeester de Monchyplein, The Hague, Netherlands, Dr. L. Hoedemaker, secretary to the organizing committee.

Eighth International Congress of Animal Husbandry. Hamburg, Germany, June 13, 1961.

First International Congress of Endocrinology. Technical University of Denmark, Copenhagen, July 18-23, 1960. Dr. Christian Hamburger, Statens Seruminstitut, Copenhagen S, Denmark, chairman of the executive committee.

Regularly Scheduled Meetings

ALABAMA—Central Alabama Veterinary Medical Association, the first Thursday of each month. James L. Chambers, 4307 Normanbridge Rd., Montgomery, Ala., secretary-treasurer.

Jefferson County Veterinary Medical Association, the second Thursday of each month. Dan P. Griswold, Jr., 714 S. 39th St., Birmingham, secretary.

Mobile-Baldwin Veterinary Medical Association, the third Tuesday of each month. Cecil S. Yarbrough, 4121 U.S. 90 West, Mobile, Ala., secretary.

Northeast Alabama Veterinary Medical Association, the second Tuesday of every other month. Leonard J. Hill, P.O. Box 761, Gadsden, Ala., secretary-treasurer.

ALASKA—Anchorage Group of the Alaska V. M. A., the last Wednesday of each month at Fort Richardson Officers' Club or Thompson's Restaurant, 6th and I Streets, Anchorage, Alaska. Lt. Colonel E. H. Akins, Surgeon's Office, USARAL, Fort Richardson, Alaska, secretary to the Alaska V. M. A.

Treatment of Monkeys

Tuberculosis, pneumonia, and dysentery are probably the most prevalent monkey diseases. Tuberculosis is best controlled by test and eradication. The intradermal palpebral test with radiographs of the thorax are excellent aids to tuberculosis diagnosis. Retest in 30 days may show as false negatives.

Monkeys with respiratory diseases should be treated promptly with penicillin, other antibiotics, sulfonamides, and general supportive therapy. Increased room temperature and supplementation with energy foods like Karo syrup and honey are advisable.

Intestinal diseases should be treated with diet control, adequate fluids, and use of drugs like streptomycin, neomycin, nitrofurazone, and astringents (bismuth, kaolin, pectin, and carob powder preparations).

Strongyloides are frequently encountered intestinal parasites along with ascarids and tapeworms. Piperazine, phenothiazine, and other vermifuges like gentian violet and hexylresorcinol are effective. Oxytetracycline is useful in treatment of amebic dysentery.—*L. E. Fisher at the Symposium on Health of Small Animals, Chicago, July, 1959.*

ARIZONA—Central Arizona Veterinary Medical Association, the second Tuesday of each month. John D. Clark, 518 West Oak St., Scottsdale, Ariz., secretary.

Southern Arizona Veterinary Medical Association, the third Wednesday of each month at 7:30 p.m. Gwyn Chapin, 2215 E. Calle Vista, Tuscon, Ariz., secretary.

ARKANSAS—Pulaski County Veterinary Medical Society, the second Tuesday of each month. Harvie R. Ellis, 54 Belmont Drive, Little Rock, Ark., secretary-treasurer.

CALIFORNIA—Alameda-Contra Costa Veterinary Medical Association, the fourth Wednesday of Jan., March, May, June, Aug., Oct., and Nov. John S. Blackard, 420 Appian Way, Richmond, Calif., secretary.

Bay Counties Veterinary Medical Association, the second Tuesday of February, April, July, September, and December. Herb Warren, 3004 16th St., San Francisco, Calif., executive secretary.

Central California Veterinary Medical Association, the fourth Tuesday of each month. Paul S. Chaffee, 2333 McKinley Ave., Fresno, Calif., secretary.

Humboldt-Del Norte Counties Veterinary Medical Association, the second Tuesday of January, May, September, and November. Dr. M. Lunstra, P. O. Box 734, Eureka, Calif., secretary-treasurer.

Kern County Veterinary Medical Association, the first Thursday evening of the month. Francis Dawson, 2007 Nile St., Bakersfield, Calif., secretary-treasurer.

Mid-Coast Veterinary Medical Association, the first Thursday of each month. William P. Matulich, P. O. Box 121, San Luis Obispo, Calif., secretary-treasurer.

Monterey Bay Area Veterinary Medical Association, the third Wednesday of each month. V. Todorovic, 47 Mann Ave., Watsonville, Calif., secretary.

Northern California Association of Veterinarians, the second Tuesday of the month. George Crenshaw, 1137 8th St., Orland, Calif., secretary.

North San Joaquin Valley Veterinary Medical Association, the fourth Wednesday of each month at the Hotel Covell in Modesto, Calif. Kenneth E. Erwin, Box 841, Manteca, Calif., secretary.

Orange Belt Veterinary Medical Association, the second Monday of each month. M. C. McSpadden, 2372 West Ramsey, Banning, Calif., secretary.

Orange County Veterinary Medical Association, the third Thursday of each month. H. M. Stanton, 1122 S.E. U.S. Highway 101, Tustin, Calif., secretary.

Peninsula Veterinary Medical Association, the third Monday of the month. R. M. Grandfield, 416 Stephens Rd., San Mateo, Calif., secretary-treasurer.

Redwood Empire Veterinary Medical Association, the third Thursday of the month. R. R. Rediske, 833 Vallejo Ave., Novato, Calif., secretary-treasurer.

Sacramento Valley Veterinary Medical Association, the second Thursday of each month with the location specified monthly. Eugene C. Story, 4819 "V" St., Sacramento 17, Calif., secretary.

San Diego County Veterinary Medical Association, the fourth Tuesday of the month. Robert F. Burns, 7572 North Ave., Lemon Grove, Calif., secretary-treasurer.

San Fernando Valley Chapter SCVMA, the second Tuesday of each month at 7:30 p.m., Hody's Restaurant, North Hollywood, Calif. Barbara G. Shirley, Canoga Park, Calif., secretary-treasurer.

San Fernando Valley Veterinary Medical Association, the second Friday of each month at the Casa Escobar Restaurant in Studio City. John Chudacoff, 7912 Sepulveda Blvd., Van Nuys, Calif., secretary.

San Francisco Veterinarians, every other month—meetings decided at previous sessions. J. Wachs, 317D Sacramento St., San Francisco, Calif., secretary-treasurer.

Santa Barbara-Ventura Counties Veterinary Medical Association, every three months, no set date. Gerald M. Clark, 5415 8th St., Carpinteria, Calif., secretary-treasurer.

Santa Clara Valley Veterinary Medical Association, the last Tuesday of the month. Robert L. King, 1269 Grant St., Santa Clara, Calif., secretary-treasurer.

Southern California Veterinary Medical Association, the third Wednesday of the month. Mr. Don Mahan, 1919 Wilshire Blvd., Los Angeles 57, Calif., executive secretary.

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Tulare County Veterinary Medical Association, the second Thursday of each month at the Tagus Ranch, Tulare. Larry A. Jackson, 12191 Lacey Blvd., Hanford, Calif., secretary-treasurer.

COLORADO—Denver Area Veterinary Medical Society, the fourth Tuesday of every month. Gene M. Bierhaus, 2896 S. Federal Blvd., Englewood, Colo., secretary-treasurer.

Northern Colorado Veterinary Medical Society, the first Wednesday of each month, in Fort Collins. E. J. Carroll, Dept. of Clinics and Surgery, Colorado State University, Fort Collins, Colo., secretary.

DELAWARE—New Castle County Veterinary Medical Association, the first Tuesday of each month at 9:00 p.m. in the Hotel Rodney, Wilmington, Del. A. P. Mayer, Jr., R.F.D. 2, Newark, Del., secretary-treasurer.

DISTRICT OF COLUMBIA—District of Columbia Veterinary Medical Association, the second Tuesday evenings of January, March, May, and October. R. B. Gochenour, 10109 Ashwood Dr., Kensington, Md., secretary-treasurer.

FLORIDA—Big Bend Veterinary Medical Association, meets the first Sunday of each month at 5:00 p.m. at the Tallahassee Dining Room, Tallahassee. C. Paul Vickers, P.O. Box 309, Talahassee, secretary.

Central Florida Veterinary Medical Association, the first Friday of each month at 8:00 p.m., place specified monthly. L. R. Poe, 753 W. Fairbanks Ave., Winter Park, Fla., secretary-treasurer.

Florida West Coast Veterinary Medical Association, the second Wednesday of each month at the Lighthouse Inn, St. Petersburg. Fred Jones, 3606 S. Dale Mabry, Tampa, Fla., secretary.

Hillsborough Veterinary Medical Society, the second Monday of every month; time and place are specified monthly. J. H. Rogers, 311 N. Ben Avon Dr., Tampa, Fla., secretary-treasurer.

Jacksonville Veterinary Medical Association, the first Thursday of every month at the Green Turtle Restaurant, at 8:00 p. m. Edwin G. Clampett, 5150 Love Grove Rd., Jacksonville 7, Fla., secretary.

Northwest Florida Veterinary Medical Society, third Wednesday of each month, time and place specified monthly. John Webb, P.O. Box 183, Cantonment, Fla., secretary-treasurer.

Palm Beach Veterinary Society, the last Thursday evening of each month. McArthur Dairy Building, Four Points, W. Palm Beach. B. W. Bigger, 2833 S. 4th St., Fort Pierce, Fla., secretary.

Pinellas County Veterinary Society, the first Monday of January, February, April, May, July, August, October, and November at the Fort Harrison Hotel, Clearwater, Fla., at 7:30 p.m. L. H. Sellers, Jr., 3813 Tyrone Blvd., St Petersburg, Fla., secretary.

Ridge Veterinary Medical Association, the fourth Thursday of each month in Bartow, Fla. John S. Haromy, Route #1, Box 107-A, Lake Wales, Fla., secretary.

South Florida Veterinary Society, the third Wednesday of each month. Time and place specified monthly. Joe B. O'Quinn, 1690 E. 4th, Hialeah, Fla., secretary.

Suwanee Valley Veterinary Association, the fourth Tuesday of each month, Hotel Thomas, Gainesville. G. L. Burch, P.O. Box 405, Ocala, Fla., secretary-treasurer.

Volusia County Veterinary Medical Association, the fourth Thursday of each month. Robert E. Cope, 127 E. Mason, Daytona Beach, Fla., secretary.

GEORGIA—Atlanta Veterinary Medical Society, the third Thursday of each month at the Elk's Home, 726 Peachtree St., Atlanta. Clare L. Bromley, 634 Northside Dr., N.W., Atlanta, Ga., secretary.

Georgia-Carolina Veterinary Medical Association, the second Monday of each month at 8:00 p.m., at the Town Tavern, Augusta, Ga. J. A. Schmitz, 1711 Gwinnett St., Augusta, Ga., secretary.

North Georgia Veterinary Medical Association, quarterly, no set date, the spring meeting at the Veterinary School, Athens, Ga. S. J. Shirley, Commerce, Ga., secretary.

Southeast Georgia Veterinary Medical Association, quarterly, date and meeting place varies. Hugh F. Arundel, P.O. Box 153, Statesboro, Ga., secretary.

South Georgia Veterinary Medical Association, the second Sunday of each quarter at 3:30 p.m., at the Radium Springs Hotel, Albany, Ga. M. W. Hale, Route 2, Tifton, Ga., secretary.

ILLINOIS—Chicago Veterinary Medical Association, the second Tuesday of each month. Charles H. Armstrong, 1021 Davis St., Evanston, secretary.

INDIANA—Calumet Area Veterinary Medical Association, the first Thursday of each month. Bruce Sharp, Box 166, Hobart, Ind., secretary-treasurer.

Central Indiana Veterinary Medical Association, the second Wednesday of each month. P. T. Parker, 224 N. Mill St., Plainfield, Ind., secretary-treasurer.

Michiana Veterinary Medical Association, the second Thursday of every month except July and December, at the Hotel LaSalle, South Bend, Ind. Stanton Williamson, 217 W. Chippewa St., South Bend, Ind., secretary.

Northwestern Indiana Veterinary Medical Association, the fourth Thursday of each month, except August, January, and February. Harvey R. Smith, R.R. 2, Box 30, Lowell, Ind., secretary-treasurer.

Tenth District Veterinary Medical Association, the third Thursday of each month. J. S. Baker, P.O. Box 52, Pendleton, Ind., secretary.

IOWA—Cedar Valley Veterinary Medical Association, the second Monday of each month, except January, July, August, and October in Black's Tea Room, Waterloo, Iowa. A. J. Cotten, P.O. Box 183, Grundy Center, secretary.

Central Iowa Veterinary Medical Association, the third Monday of each month except June, July, and August at 6:30 p.m., Breeze House, Ankeny, Iowa. S. L. Hendricks, secretary-treasurer.

Coon Valley Veterinary Medical Association, the second Wednesday of each month, September through May, at 7:30 p.m., Cobblestone Inn, Storm Lake, Iowa. Robert McCutcheon, Holstein, secretary.

East Central Iowa Veterinary Medical Society, the second Thursday of each month at 6:30 p.m., usually in Cedar Rapids, Iowa. T. F. Bartley, P.O. Box 454, Cedar Rapids, secretary.

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Fayette County Veterinary Medical Association, the third Thursday of each month at 6:30 p.m. in West Union, Iowa. H. J. Morgan, West Union, secretary.

Lakes Veterinary Association, the first Tuesday of each month, September through May, at 6:30 p.m., at the Gardson Hotel, Estherville, Iowa. Barry Barnes, P.O. Box 162, Milford, secretary.

North Central Iowa Veterinary Medical Association, the third Thursday of April, at the Warden Hotel, Fort Dodge, Iowa. H. Engelbrecht, P. O. Box 797, Fort Dodge, secretary.

Northeast Iowa-Southern Minnesota Veterinary Association, the first Tuesday of February, May, August, and November at the Wisneslick Hotel, Decorah, Iowa, 6:30 p.m. Donald E. Moore, Box 178, Decorah, Iowa, secretary.

Northwest Iowa Veterinary Medical Association, the second Tuesday of February, May, September, and December, at the Community Bldg., Sheldon. W. Ver Meer Hull, secretary.

Southeastern Iowa Veterinary Association, the first Tuesday of each month at Mt. Pleasant, Iowa. Warren Kilpatrick, Mediapolis, secretary.

Southwestern Iowa Veterinary Medical Association, the first Tuesday of April and October, Hotel Chieftain, Council Bluffs, Iowa. J. P. Stream, 202 S. Stone St., Creston, secretary.

KENTUCKY—Central Kentucky Veterinary Medical Association, the first Wednesday of each month. R. H. Folsom, P.O. Box 323, Danville, Ky., secretary.

Jefferson County Veterinary Society of Kentucky, Inc., the first Wednesday of each month in Louisville or within a radius of 50 miles, except January, May, and July. G. R. Comfort, 2102 Reynolds Lane, Louisville, Ky., secretary-treasurer.

LOUISIANA—New Orleans Veterinary Medical Association, the third Thursday of every month at the Monte leone Hotel, New Orleans, at 8:30 p.m. Ronald C. Francis, 6421 Chef Menteur Highway, New Orleans, La., secretary-treasurer.

MARYLAND—Baltimore City Veterinary Medical Association, the second Thursday of each month, September through May (except December), at 9:00 p.m., at the Park Plaza Hotel, Charles and Madison St., Baltimore, Md. Leonard D. Krinsky, 6111 Hartford Rd., Baltimore, Md., secretary.

MICHIGAN—Central Michigan Veterinary Medical Association, the first Wednesday of every month at 7 p.m. Jerry Fries, 2070 E. Main St., Owosso, Mich., secretary.

Mid-State Veterinary Medical Association, the fourth Thursday of each month with the exception of November and December. Robert W. Acton, 4110 Spring Rd., Jackson, Mich.

Saginaw Valley Veterinary Medical Association, the last Wednesday of each month. Alvin R. Conquest, P.O. Box 514, Grand Blanc, Mich., secretary.

Southeastern Michigan Veterinary Medical Association, the fourth Wednesday of every month, September through May. Louis J. Rossoni, 24531 Princeton Ave., Dearborn 8, Mich., secretary.

MINNESOTA—Lake Region Veterinary Medical Association, quarterly meetings, with time and place specified prior to each meeting. J. A. Strache, Battle Lake, Minn., secretary-treasurer.

MISSOURI—Greater St. Louis Veterinary Medical Association, the first Friday of each month (except July and August), at the Coronado Hotel, Lindell Blvd. and Spring Ave., St. Louis Mo., at 8 p.m. Edwin E. Epstein, 4877 Natural Bridge Ave., St. Louis 15, Mo., secretary.

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Kansas City Veterinary Medical Association and Kansas City Small Animal Hospital Association, the third Thursday of each month at the Hotel President, Kansas City, Mo. Robert E. Guilfoil, 18 N. 2nd St., Kansas City 18, Kan., secretary.

NEVADA—Western Nevada Veterinary Society, the first Tuesday of each month. Paul S. Silva, 1170 Airport Road, Reno, Nev., secretary.

NEW JERSEY—Central New Jersey Veterinary Medical Association, the second Thursday of November, January, March, and May at Old Hights Inn, Hightstown, N. J. David C. Tudor, R.D. 1, Box 284A, Cranbury, N. J., secretary.

Metropolitan New Jersey Veterinary Medical Association, the third Wednesday evening of each month from October through April, except December, at the Irvington House, 925 Springfield Ave., Irvington, N.J. Bernard M. Weiner, 787 Clinton Ave., Newark, N.J., secretary.

Northern New Jersey Veterinary Association, the fourth Tuesday of each month at the Elks Club, Hackensack. James R. Tanzola, Upper Saddle River, N.J., secretary.

Northwest Jersey Veterinary Society, the third Wednesday of every odd month. G. L. Smith, P.O. Box 938, Trenton, N.J., secretary.

South New Jersey Veterinary Medical Association, the fourth Tuesday of each month at the Collmont Diner, Collingswood, N.J. Marvin Rothman, 718 Dwight Ave., Collingswood, N. J., secretary.

NEW MEXICO—Bernalillo County Veterinary Practitioners Association, the third Wednesday of each month, Fez Club, Albuquerque. Donald W. Fitzgerald, 1825 Lomas Blvd., N.E., Albuquerque, N.M., secretary-treasurer.

NEW YORK—New York City, Inc., Veterinary Medical Association of, the first Wednesday of each month at the New York Academy of Sciences, 2 East 63rd St., New York City. C. E. DeCamp, 43 West 61st St., New York 23, N. Y., secretary.

Monroe County Veterinary Medical Association, the first Thursday of even-numbered months except August. Irwin Bircher, 50 University Ave., Rochester, N. Y., secretary.

NORTH CAROLINA—Central Carolina Veterinary Medical Association, the second Wednesday of each month at 7:00 p.m. in the O'Henry Hotel, Greensboro. W. A. Sumner, 3741 High Point Rd., Greensboro, N.C., secretary.

Eastern North Carolina Veterinary Medical Association, the last Tuesday evening of each month, time and place specified monthly. Byron H. Brow, Box 453, Goldsboro, N. Car., secretary-treasurer.

Piedmont Veterinary Medical Association, the last Friday of each month. J. G. Martin, Boone, N. Car., secretary.

Twin Carolinas Veterinary Medical Association, the third Friday of each month at Orange Bowl Restaurant, Rockingham, N. Car., at 7:30 p.m. J. E. Currie, 690 N. Leak St., Southern Pines, N. Car., secretary.

Western North Carolina Veterinary Medical Association, the third Thursday of every month at 7:00 p.m. in the George Vanderbilt Hotel, Asheville, N. Car. H. A. Justus, 924 Lakeside Dr., Hendersonville, N. Car., secretary.

OHIO—Cincinnati Veterinary Medical Association, the third Tuesday of every month at Shuller's Wigwam, 6210 Hamilton Ave., at North Bend Road. W. H. Shartle, 323 S. Front St., Hamilton, Ohio, secretary.

Clark County Veterinary Medical Association, meetings held quarterly; time and place irregular. R. Edmondson, South Charleston, Ohio, secretary-treasurer.

Columbus Academy of Veterinary Medicine, the third Thursday of every month, September through May; place irregular. R. W. Vesper, 1481 Doten Ave., Columbus, Ohio, secretary-treasurer.

Cuyahoga County Veterinary Medical Association, the first Wednesday in September, October, December, February, March, April, and May, at 9:00 p.m., at the Carter Hotel, Cleveland, Ohio. R. W. Stockstill, 6545 Mayfield Rd., Cleveland, Ohio, secretary.

Dayton Veterinary Medical Association, the third Tuesday of every month; place irregular. William Pumpelly, 6801 Airway Rd., Dayton, Ohio, secretary-treasurer.

Fifth District Veterinary Medical Association, meetings held quarterly; time and place irregular. E. J. Kersting, 115 Sheffield Rd., Columbus, Ohio, secretary.

Geauga County Veterinary Medical Society, the third Wednesday of each month, at the Manor House, Newberry, Ohio, at 1:00 p.m. Peter J. Clemens, Jr., R. D. 2, Chagrin Falls, Ohio, secretary.

Kilbuck Valley Veterinary Medical Association, the first Wednesday of alternate months beginnig with February; place irregular. Charles Gale, Ohio Agricultural Experiment Station, Wooster, Ohio, secretary-treasurer.

Knox County Veterinary Medical Association, meetings irregular. F. O. Haberman, Centerburg, Ohio, president.

Lorain County Veterinary Medical Association the second Wednesday of April, June, October, December, and February; place irregular. G. W. Bunyan, 37200 Detroit Rd., Avon, Ohio, secretary-treasurer.

Madison County Veterinary Medical Association, quarterly; date and place irregular. James Herman, Mechanicsburg, Ohio, secretary-treasurer.

Mahoning County Veterinary Medical Association, the fourth Tuesday of each month, at 9:00 p.m. Youngstown Maennerchor Club, Youngstown, Ohio. Robert Edwards, 25 Oriole Dr., Youngstown, Ohio.

Miami Valley Veterinary Medical Association, the first Wednesday of December, March, June, and September; place iregular. L. J. Grilliot, Route No. 1, Troy, Ohio, secretary-treasurer.

North Central Ohio Veterinary Medical Association, the last Wednesday of each month, except during summer months; place irregular. Ben Henson, 268 S. Main St., Mansfield, Ohio, secretary-treasurer.

Northwestern, Ohio Veterinary Medical Association the last Wednesday of March and July; place irregular. F. C. Hartman, 3904 Rushland Ave., Toledo, Ohio, secretary-treasurer.

Southeastern Ohio Academy of Veterinary Medicine, every other month; time and place irregular. M. S. Phillips, Athens, Ohio, secretary.

Southern Ohio Veterinary Medical Association, meetings held quarterly at Wilmington; time irregular, S. E. Peterson, 1093 Rombach Ave., Wilmington, Ohio, secretary-treasurer.

Stark County Veterinary Medical Association, the second Tuesday of every month, at McBride's Emerald Lounge, Canton. Robert Leed, 5500 Cleveland Ave., N. W., North Canton, Ohio, secretary-treasurer.

Summit County Veterinary Medical Association, the last Tuesday of every month, excepting June, July, and August, at the Mayflower Hotel, Akron. M. L. Scott, 42 W. Market St., Akron, Ohio, secretary-treasurer.

Toledo Veterinary Medical Association, every other month; date and place irregular. F. C. Hartman, 3904 Rushland Ave., Toledo, Ohio, corresponding secretary.

Trumbull County Veterinary Medical Association, meet three or four times a year; time and place irregular. R. A. Hanawalt, P.O. Box 117, Kinsman, Ohio, secretary-treasurer.

West Central Veterinary Medical Association, third Thursday of February, June, September, and November, at the Lima Club, Lima. K. R. Heidt, 1055 Spencerville Rd., Lima, Ohio, secretary-treasurer.

Wheeling Valley Veterinary Medical Association, meetings held quarterly; time and place irregular. Earl Weaver, 1406 S. Zane Rd., Martins Ferry, Ohio, vice-president.

Miami Valley Veterinary Medical Association, the first Wednesday of December, March, June, and September. J. M. Westfall, Greenville, Ohio, secretary-treasurer.

North Central Ohio Veterinary Medical Association, the last Wednesday of each month except during the summer. R. W. McClung, Tiffin, Ohio, secretary-treasurer.

Northwestern Ohio Veterinary Medical Association, the last Wednesday of March and July. C. S. Alvanos, 1683 W. Bancroft St., Toledo, Ohio, secretary-treasurer.

Stark County Veterinary Medical Association, the second Tuesday of every month, at McBrides Emerald Lounge, Canton, Ohio. M. L. Willen, 4423 Tuscarawas St., Canton, Ohio, secretary.

Summit County Veterinary Medical Association, the last Tuesday of every month (except June, July, and August), at the Mayflower Hotel, Akron, Ohio. M. L. Scott, 42 W. Market St., Akron, Ohio, secretary-treasurer.

Tri-County Veterinary Medical Association, the fourth Wednesday of January, May, and September. Mrs. R. Slusher, Mason, Ohio, secretary-treasurer.

OKLAHOMA—Oklahoma County Veterinary Medical Association, the second Wednesday of every month, 7:30 p.m., Patrick's Foods Cafe, 1016 N.W. 23rd St., Oklahoma City. Claude A. Tigert, 3032 N.W. 68th St., Oklahoma City, Okla., secretary.

Tulsa Veterinary Medical Association, the third Thursday of each month at the City-County Health Building,

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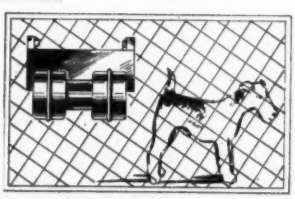

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4616 E. 15th St., Tulsa, Okla. Arlen D. Hill, 5302 E. 11th St., Tulsa, Okla., secretary.

Tulsa Association of Small Animal Veterinarians, first and third Mondays. City-County Health Dept. R. H. Featherston, 3129 S. Winston, Tulsa 5, Okla., secretary.

OREGON—Portland Veterinary Medical Association, the second Tuesday of each month, at 7:30 p.m. Ireland's Restaurant, Lloyds, 718 N.E. 12th Ave., Portland. Donald L. Moyer, 8415 S.E. McLoughlin Blvd., Portland 2, Ore., secretary.

Willamette Veterinary Medical Association, the third Tuesday of each month, except July and August, at the Marion Hotel, Salem. Robert J. Mallorie, P.O. Box 155, Silverton, Ore., secretary.

PENNSYLVANIA—Keystone Veterinary Medical Association, the fourth Wednesday of each month at the University of Pennsylvania School of Veterinary Medicine. Raymond C. Snyder, N.E. Corner 47th St. and Hazel Ave., Philadelphia 43, Pa., secretary.

Lehigh Valley Veterinary Medical Association, the first Thursday of each month. Stewart Rockwell, 10th and Chestnut Sts., Emmaus, Pa., secretary.

You can do **two things** to guard yourself against cancer: Have an annual health checkup. Alert yourself to the seven danger signals that could mean cancer: **1.** Unusual bleeding or discharge. **2.** A lump or thickening in the breast or elsewhere. **3.** A sore that does not heal. **4.** Change in bowel or bladder habits. **5.** Hoarseness or cough. **6.** Indigestion or difficulty in swallowing. **7.** Change in a wart or mole. If your signal lasts longer than two weeks, go to your physician. Give him the chance to give you the chance of a lifetime.

AMERICAN CANCER SOCIETY

Pennsylvania Northern Tier Veterinary Medical Association, the third Wednesday of each odd numbered month. R. L. Michel, Troy, Pa., secretary.

SOUTH CAROLINA—Piedmont Veterinary Medical Association, the third Wednesday of each month at the Fairforest Hotel, Union, S. Car. Worth Lanier, York, S. Car., secretary.

Georgia-Carolina Veterinary Medical Association—see GEORGIA.

TEXAS—Coastal Bend Veterinary Association, the second Wednesday of each month. Jack E. Habluetzel, Route 1, Box 65-N, Ingleside, Texas, secretary.

Dallas County Veterinary Medical Association, the first Tuesday of each month at 7:30 p.m., at a place to be specified. Frank N. Black, 12830 Preston Rd., Dallas, Texas, corresponding secretary.

UTAH—Salt Lake Small Animal Hospital Association, the first Monday of every month, at the Holiday Inn, 3040 South State St., Salt Lake City, at 12:15 p.m. Douglas H. McKelvie, 1220 S. State St., Salt Lake City, Utah, secretary-treasurer.

VIRGINIA—Central Virginia Veterinary Association, the second Thursday of each month at 8:00 p.m., except July and August, at a place in Richmond to be announced monthly. Edwin M. Crawford, secretary-treasurer.

Northern Virginia Veterinary Conference Association, the second Tuesday of each month. T. P. Koudelka, P.O. Box 694, Harrisonburg, Va., secretary.

Northern Virginia Veterinary Society, the second Wednesday of every third month. Meeting place announced by letter. H. C. Newman, Box 145, Merrifield, secretary.

Southwest Virginia Veterinary Medical Association, the first Thursday of each month. D. F. Watson, Blacksburg, secretary.

WASHINGTON—Seattle Veterinary Medical Association, the third Monday of each month, Magnolia American Legion Hall, 2870 32nd W., Seattle. Roy C. Toole, 10415 Main St., Bellevue, secretary.

South Puget Sound Veterinary Association, the second Thursday of each month except July and August. B. D. Benedictson, 3712 Plummer St., Olympia, Wash., secretary.

WEST VIRGINIA—Kyowva (Ky., Ohio, W. Va.) Veterinary Medical Association, the third Thursday of each month in the Hotel Pritchard, Huntington, W. Va., at 8:30 p.m. Harry J. Fallon, 200 5th St., W. Huntington, W. Va., secretary.

WISCONSIN—Central Wisconsin Veterinary Medical Association, the second Tuesday of each quarter (March, June, Sept., Dec.) C. R. Carlson, 1109 E. LaSalle Ave., Barron, Wis., secretary.

Coulee Region Veterinary Medical Association, the third Wednesday of every other month. F. N. Petersen, Box 127, Cashton, Wis., secretary.

Dane County Veterinary Medical Association, the second Thursday of each month. All but the special meetings will be held at the Top Hat Restaurant, Middleton. Robert E. Hall, 5718 Dogwood Place, Madison 5, Wis., secretary-treasurer.

Milwaukee Veterinary Medical Association, the third Tuesday of each month, at the Half-Way House, Blue Mound Rd. Dr. Raymond Pahle, 10827 W. Oklahoma Ave., Milwaukee, Wis.

Northeastern Wisconsin Veterinary Medical Association,

the third Wednesday in April. William Madson, 218 E. Washington St., Appleton, Wis., secretary.

Rock Valley Veterinary Medical Association, the first Wednesday of each month. L. C. Allenstein, 209 S. Taft St., Whitewater, Wis., secretary.

Southeastern Veterinary Medical Association, the third Thursday of each month. John R. Curtis, 419 Cook St., Portage, Wis., secretary.

Wisconsin Valley Veterinary Medical Association, the second Tuesday of every other month. John B. Fleming, 209 E. 4th St., Marshfield, Wis., secretary.

Wins Dogdom's Nobel Award

A 5-year-old mongrel named "Ruff," chosen from among 9 canine entrants for "Research Dog of the Year," was awarded a silver collar during ceremonies at the New York Academy of Sciences Dec. 12, 1959.

Fastening the chain around his neck are Elizabeth De Pasqua, 10 (left), and Laurie Ann Cloke, 4, whose lives were saved by open heart surgery made possible by experimentation with thousands of dogs like Ruff. The award, the canine version of the Nobel Prize, was presented to the brown 30-lb mongrel for his aid to medical research. Ruff, once destined for execution in a public pound, is probably the best equipped dog in the world to withstand a heart attack. He's got a "booster heart" to help in case the one he was born with falters. Prize was awarded by National Society for Medical Research.

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Illustrations.—Photographs should be furnished in glossy prints, and of a size that will fit into the JOURNAL of the American Veterinary Medical Association with a minimum of reduction. Photomicrographs which can not be reduced should be marked for cropping to 1-column or 2-column width. Identifying marks within the photomicrographs, such as arrows, letters, or numbers, should be clearly marked with black India ink or white opaque ink to insure good contrast for reproduction and must be large enough to stand reduction, if necessary.

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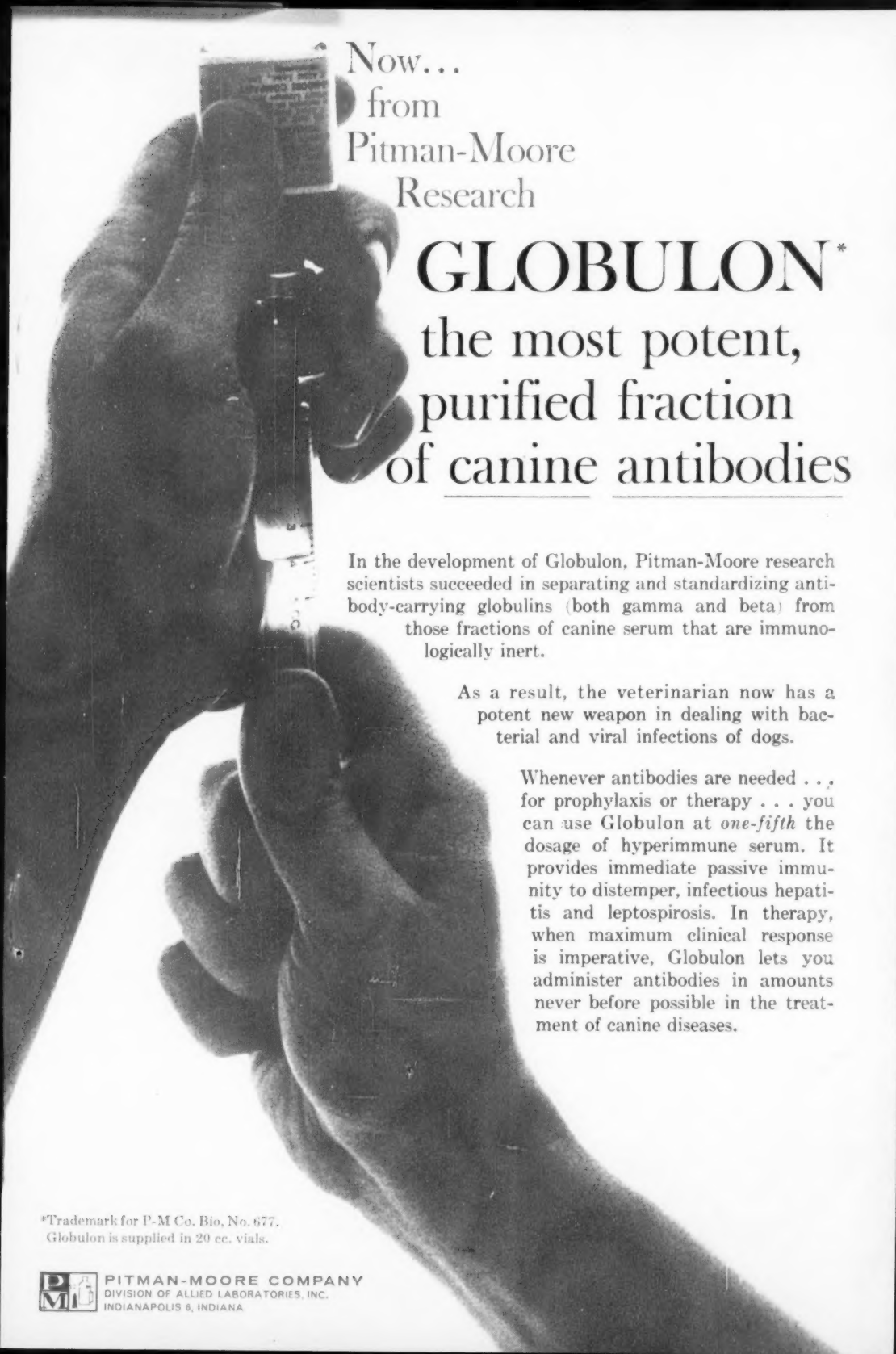

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